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Health and Environmental Effects Profile for Antimony Oxides

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16. ABSTRACT

The Health and Environmental Effects Profile for antimony oxides was prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste and Emergency Response to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA) and to provide health-related limits for emergency actions under Section 101 of the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA). Both published literature and information obtained from Agency program office files were evaluated as they pertained to potential human health, aquatic life and environmental effects of hazardous waste constituents.

Quantitative estimates have been presented provided sufficient data are available. Antimony oxides have been determined to be a systemic toxicant. An Acceptable Daily Intake (ADI), defined as the amount of a chemical to which humans can be exposed on a daily basis over an extended period of time (usually a lifetime) without suffering a deleterious effect, for antimony, antimony trioxide, antimony tetroxide and antimony pentoxide is 24.5, 29.3, 30.9 and 32.5 μg/day, respectively, for oral exposure.

The Reportable Quantity (RQ) value of 1, 10, 100, 1000 or 5000 pounds is used to determine the quantity of a hazardous substance for which notification is required in the event of a release as specified by CERCLA based on chronic toxicity. The RQ value for antimony oxides is 100.

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HEALTH AND ENVIRONMENTAL EFFECTS PROFILE FOR ANTIMONY OXIDES

ENVIRONMENTAL CRITERIA AND ASSESSMENT OFFICE
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PREFACE

Health and Environmental Effects Profiles (HEEPs) are prepared for the Office of Solid Waste and Emergency Response by the Office of Health and Environmental Assessment. The HEEPs are intended to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA), as well as to provide health-related limits for emergency actions under Section 101 of the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA). Both published literature and information obtained from Agency program office files are evaluated as they pertain to potential human health, aquatic life and environmental effects of hazardous waste constituents. The literature searched and the dates of the searches are included in the section titled "Appendix: Literature Searched." The literature search material is current through November, 1984.

Quantitative estimates are presented provided sufficient data are available. For systemic toxicants, these include Acceptable Daily Intakes (ADIs) for chronic exposures. An ADI is defined as the amount of a chemical to which humans can be exposed on a daily basis over an extended period of time (usually a lifetime) without suffering a deleterious effect. In the case of suspected carcinogens, ADIs are not estimated in this document series. Instead, a carcinogenic potency factor of q_1^* (U.S. EPA, 1980) is provided. These potency estimates are derived for both oral and inhalation exposures where possible. In addition, unit risk estimates for air and drinking water are presented based on inhalation and oral data, respectively.

Reportable quantities (RQs) based on both chronic toxicity and carcinogenicity are derived. The RQ is used to determine the quantity of a hazardous substance for which notification is required in the event of a release as specified under CERCLA. These two RQs (chronic toxicity and carcinogenicity) represent two of six scores developed (the remaining four reflect ignitability, reactivity, aquatic toxicity and acute mammalian toxicity).

The first draft of this document was prepared by Syracuse Research Corporation under EPA Contract No. 68-03-3228. The document was subsequently revised after reviews by staff within the Office of Health and Environmental Assessment: Carcinogen Assessment Group, Reproductive Effects Assessment Group, Exposure Assessment Group, and the Environmental Criteria and Assessment Office in Cincinnati.

The HEEPs will become part of the EPA RCRA and CERCLA dockets.

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LIST OF ABBREVIATIONS

ADI	Acceptable daily intake
AOIA	Antimony Oxide Industry Association
BCF	Bioconcentration factor
bw	Body weight
CAS	Chemical Abstracts Service
CS	Composite score
DNA	Deoxyribonucleic acid
EC_{50}	Median effective concentration
EF	Enrichment factor
EKG	Electrocardiogram
ESP	Electrostatic precipitators
GI	Gastrointestinal
i.p.	Intraperitoneal
i.v.	Intravenous
LC_{50}	Concentration lethal to 50% of recipients
LD_{50}	Dose lethal to 50% of recipients
LOEL	Lowest observed effect level
MATC	Maximum acceptable toxicant concentration
MED	Minimum effective dose
NOEC	No observed effect concentration
ppb	Parts per billion
ppm	Parts per million
ppt	Parts per trillion
RQ	Reportable quantity
RV_d	Dose-rating value
RV_e	Effect-rating value
s.c.	Subcutaneous
TLV	Threshold limit value
UV	Ultraviolet
W	Washout factor

1. INTRODUCTION

1.1. STRUCTURE AND CAS NUMBER

Antimony trioxide is the common name for the compound with the chemical formula Sb_2O_3. It is also known by several synonyms, including antimony white, antimony oxide, antimonious oxide, antimony peroxide, antimony sesquioxide, diantimony trioxide, exitelite, senarmontite and valentinite. Trade names for this compound include Thermoguard S, White Star, Stibnox, Montana Brand, Fire Shield H, C.I. Pigment White 11 (Avento and Touval, 1978) and Stibnox (Carapella, 1978).

The chemical formula for antimony trioxide is Sb_2O_3 and the formula weight is 291.52. The CAS Registry number for antimony trioxide is 1309-64-4. The CAS Registry number for antimony oxide (understood to be Sb_2O_3) is 1327-33-9. The CAS Registry numbers for the two naturally-occurring forms are senarmontite, 12412-52-1; and valentinite, 1317-98-2.

Antimony tetroxide is the common name for the compound with the chemical formula Sb_2O_4. It is also known as antimony dioxide, antimony (III,V) oxide, cervantite or α-antimony tetroxide, and β-antimony tetroxide.

The chemical formula for antimony tetroxide is Sb_2O_4 (or $Sb_2O_3 \cdot Sb_2O_5$) and the formula weight is 307.50. The CAS Registry number for this compound is 1332-81-6.

Antimony pentoxide is the common name for the compound with the chemical formula Sb_2O_5. It is also known as antimonic acid, antimony(V) oxide, antimonic(V) acid, antimonic anhydrile and stibic anhydride.

The chemical formula for antimony pentoxide is SB_2O_5 (or Sb_4O_{10}) and the formula weight is 323.52. The CAS Registry number for this compound is 1314-60-9. Antimony pentoxide is always found in hydrated form (Stewart et al., 1972; Freedman et al., 1978). The CAS Registry number for antimony pentoxide pentahydrate is 12534-39-3.

1.2. PHYSICAL AND CHEMICAL PROPERTIES

Antimony trioxide is a white, odorless, crystalline solid at room temperature (Hawley, 1977). It occurs in two crystalline modifications, orthorhombic (natural valentinite) and cubic (natural senarmontite) (Freedman et al., 1978). Some of the physical properties of Sb_2O_3 are listed in Table 1-1.

Antimony tetroxide is a white, crystalline solid at room temperature (Weast, 1973). It occurs in two crystalline modifications, orthorhombic (natural cervantite, α-antimony tetroxide) and monoclinic (β-antimony tetroxide). Antimony tetroxide is a mixture of antimony (III) oxide and antimony (V) oxide (i.e., chemical formula: $Sb_2O_3 \cdot Sb_2O_5$) (Stewart et al., 1972). Some of the physical properties of antimony tetroxide are listed in Table 1-1.

Antimony pentoxide is a yellowish, crystalline solid that is always somewhat hydrated (Windholz, 1976). It has been stated that there is no satisfactory evidence for the existence of an anhydrous form of antimony pentoxide. Some of the physical properties of antimony pentoxide are listed in Table 1-1.

The U.S. EPA (1983) has issued a warning concerning the actual identity of the individual antimony substances that were the subject of the available information on the production, human exposure to and environmental release of Sb metal, Sb_2O_3 and Sb_2S_3, as well as information on the potential environmental and health effects of exposure to the antimony substances: 1) no available analytical techniques can chemically distinguish among Sb, Sb_2O_3 or Sb_2S_3 at very low levels, 2) these substances are frequently produced and used in the same facilities by the same workers, and 3) these substances are converted from one substance to another during some

TABLE 1-1

Physical Properties

Property	Antimony Trioxide	Antimony Tetroxide	Antimony Pentoxide
Empirical formula:	Sb_2O_3	Sb_2O_4	Sb_2O_5
Molecular weight:	291.52	307.50	323.52
CAS Registry number:	1309-64-4	1332-81-6	1314-60-9
Water solubility:	5 ppm (25°C)	insoluble to slightly soluble	very slightly soluble
Miscellaneous solubility:	soluble: KOH 3% HCl, concentrated HCl, concentrated H_2SO_4	soluble: strong bases, concentrated HCl; very slightly soluble: KOH, HCL, HI	very slightly soluble: KOH, HCL, HI
Melting point:	656°C	930°C (loses O)	450°C; loses O at 380°C; loses O at 930°C
Boiling point:	1425°C (870°C at 210 mm Hg	NA	NA
Vapor pressure:	1 mm HG at 574°C 6 mm Hg at 666°C	NA	NA
Specific gravity:	5.2 (senarmontite) 5.67 (valentinite)	5.82 (density)	5.6

NA = Not available

commercial and environmental processes. This has caused some overestimation of the production and exposure to individual antimony substances where these relationships were not fully considered (U.S. EPA, 1983).

1.3. PRODUCTION DATA

Antimony trioxide is produced commercially by oxidation of molten antimony trisulfide ore and/or antimony metal in air at 600-800°C (Avento and Touval, 1978), and alkaline hydrolysis of antimony trichloride followed by dehydration; it is produced directly from low-grade ores by wasting and volatilization (Hawley, 1977). Antimony tetroxide (α-modification) is formed by heating antimony trioxide in air between 460-540°C (Freedman et al., 1978). β-Sb_2O_4 is obtained either by heating α-Sb_2O_4 at 1130°C in dry air or oxygen, or by heating antimonic(V) acid ($Sb_2O_5 \cdot nH_2O$) above 900°C. At higher temperatures the solid vaporizes without first undergoing any transformation; the recondensed vapors consist of a mixture of β-Sb_2O_4 and Sb_2O_3. Antimony pentoxide is produced commercially by oxidation of antimony trioxide with nitrates or peroxides (Avento and Touval, 1978). Major manufacturers and suppliers of the antimony oxides are listed in Table 1-2 (SRI, 1984; U.S. EPA, 1984b). An additional 29 producers and suppliers of antimony trioxide were listed by the U.S. EPA (1984b). Total 1981 primary antimony oxides produced in the United States was 16,425 short tons (antimony content, 32.9 million pounds) (Plunkert, 1981). Total United States 1981 imports for consumption of antimony oxides was 12,170 short tons (antimony content, 24.3 million pounds). Total United States 1980 imports and production of antimony trioxide was 47 million pounds (39 million pounds antimony content) (Mathtech, 1981). This was 76% of the total amount of antimony oxides reportedly produced in the United

TABLE 1-2

Antimony Oxides: Manufacturers and Suppliers*

Company	Location
Antimony Trioxide:	
Anzon America Inc.	Laredo, TX
ASARCO Inc.	Omaha, NE
The Bermuth Corp.	Memphis, TN
Chemetron Corp.	LaPorte, TX
Chemetron Corp	Cleveland, OH
Cerac, Inc.	Milwaukee, WI
The Harshaw Chemical Co.	Cleveland, OH
Indussa-Div. of African Metals	New York, NY
M & T Chemicals Inc.	Baltimore, MD
Monsanto	St. Louis, MO
Monson Chemicals Inc.	Hopkinton, MA
N.L. Industries Inc.	Hightstown, NJ
N.L. Industries Inc.	Laredo, TX
Nichichem Co., Inc.-N.Y.	New York, NY
PPG Industry, Inc.	LaPorte, TX
Revelli Chemicals Inc.	Yonkers, NY
Richardson-Vicks, Inc.	Phillipsburg, NJ
Samincorp, Inc.	New York, NY
Sobin Chemicals Inc.	Boston, MA
Sumitomo Shoji America, Inc.	New York, NY
3 M Company	St. Paul, MN
Antimony Tetroxide:	
United Mineral and Chemical Corp.	New York, NY
Rhone-Poulenc, Inc.	New York, NY
Antimony Pentoxide:	
Calsicat Division	Erie, PA
Cerac, Inc.	Milwaukee, WI
Chemetron Corp.	Cleveland, OH
EM Laboratories	Westchester, NY
N. L. Industries, Inc.	Perth Amboy, NJ
Nyocol Inc.	Ashland, MA
Rhone-Poulenc, Inc.	New York, NY

*Source: SRI, 1984; U.S. EPA, 1984b

States: 15,461 short tons (antimony content, 31 million pounds) and imported: 10,148 short tons (antimony content, 20 million pounds) (Plunkert, 1981).

1.4. USE DATA

Antimony trioxide has many uses including flameproofing of textiles, paper and plastics; as a paint pigment, ceramic opacifier, catalyst, mordant and glass decolorizer; and for staining iron and phosphors (Hawley, 1977). It is used to produce antimony tetroxide and antimony pentoxide (Freedman et al., 1978) and tartar emetic (Windholz, 1976). Antimony trioxide produced from low-grade es is reduced to antimony metal (Carapella, 1978). Antimony tetroxide is used as an oxidation catalyst, particularly for oxidative dehydrogenation of olefins (Freedman et al., 1978). Antimony pentoxide is used to produce antimony tetroxide, antimonates and other antimony compounds (Hawley, 1977), and as an ion exchange material (Freedman et al., 1978).

Of the antimony trioxide consumed annually in the United States, ~90% is used as a flame retardant in engineered plastic material (M & T Chemicals, Inc., 1977). Most of the remaining 10% is used as an opacifier in cast-iron enamels or in purifying glass.

2. ENVIRONMENTAL FATE AND TRANSPORT

Discussions of available information concerning the environmental fate and transport of antimony trioxide (U.S. EPA, 1978a) and antimony, antimony trioxide and antimony trisulfide (Basu et al., 1981; Federal Register, 1983a; Newman et al., 1981) were located in the literature. Much of the available information treats antimony compounds collectively. The U.S. EPA has issued a warning concerning the actual identity of individual antimony substances, especially at very low levels, that were the subject of studies and information available in the literature (Federal Register, 1983a) (see Section 1.2.). Pertinent data regarding the fate and transport of Sb_2O_4 and Sb_2O_5 were not located in the available literature as cited in the Appendix.

2.1. AIR

Sb_2O_3 has a very low vapor pressure even at high temperatures: 1 mm Hg at 574°C and 6 mm Hg at 666°C (U.S. EPA, 1984a). Sb_2O_4 and Sb_2O_5 would also be expected to have very low vapor pressures, expecially in view of their relatively high melting (decomposition) points: Sb_2O_4, mp 930°C (loses oxygen), and Sb_2O_5 mp 380°C (loses oxygen) (Weast, 1973). Antimony oxides will not be released, therefore, by volatilization into the atmosphere at temperatures normally encountered in the environment. Substantial amounts of Sb_2O_3, however, and possibly some Sb_2O_4 and Sb_2O_5 are released to the atmosphere during processing of antimony materials including smelting of ores, molding and incineration of products, as well as the combustion of fossil fuels which all utilize the high temperatures needed to volatilize the Sb_2O_3 (Chapter 3) (Newman et al., 1981).

Furthermore, Sb_2O_4 is formed when Sb_2O_3 is heated in air between 460-540°C and when Sb_2O_5 is heated to >900°C (the recondensed vapors consist of a mixture of Sb_2O_4 and Sb_2O_3) (Freedman et al., 1978). Due to their low volatility, the antimony oxides will probably condense rapidly on suspended particulates (Bowen, 1975).

Sb_2O_3 appears to be resistant to oxidation and hydrolysis under normal environmental conditions (Basu et al., 1981). As stated above, however, oxidation of Sb_2O_3 can occur at the high temperatures sometimes encountered in the processing of antimony materials (Freedman et al., 1978).

Pertinent data regarding the photolysis in air of antimony oxides were not located in the available literature as cited in the Appendix. Since Sb_2O_3 absorbs light at wavelengths up to 325 nm (Markam et al., 1958), it is possible that some unreported photochemical processes occur

The transport and removal of antimony oxides in the atmosphere appears to depend extensively upon the condensation and adsorption of these materials on suspended particulate matter; the size distribution of these particulates is also important. Table 2-1 lists several types of manufactured Sb_2O_3 and their particle sizes. Table 2-2 lists the results of several studies of particle size distributions of anitmony particulates within combustion processes (Newman et al., 1981). Newman et al. (1981) stated that particles <1 nm in diameter are usually the most difficult to control with current emission control devices. Stokes law states that settling velocities will decrease as the particle size decreases (Perkins, 1974). The rate of removal of antimony associated particulates from the air by settling may be lower than for other metals that are normally associated with larger sized particles (Basu et al., 1981). Table 2-3 lists several of the factors which affect air residence times for Sb containing particulates and reported

TABLE 2-1

Particle Sizes of Manufactured Sb_2O_3

Name	Type/Use	Size Distribution (nm)	Reference
Fire shield	L grade	2.5-3.5	U.S. EPA, 1984a
(99.3%)	H grade	1.0-1.8	
(PPG)	Ultrafine	0.25	
NA	High pigmenting	0.8-1.8	Avento and
NA	Low pigmenting	0.9-2.5	Touval, 1978

NA = Not applicable

TABLE 2-2

Antimony Size Distributions Within Combustion Processes*

Source	Mass Median Diameter (nm)	Distribution Size Range (nm)	 Mass in Size Range (%)	Reference
Coal-fired power plant A (ESP outlet)	NA	730	1-5	Gladney et al., 1976
		14-30	2-7	
		6-14	5-12	
		2.5-6	13-20	
		1.4-2.5	13-14	
		0.7-1.4	5-9	
		0.35-0.70	4	
		<0.35	33-53	
Coal-fired power plant B (ESP inlet)	<1.0	NA	NA	Lee et al., 1975
(ESP outlet)	0.6	NA	NA	
Coal-fired power plant C (ESP outlet)	NA	<2	34-52	Gladney et al., 1978
Steel plant (no controls)	15	10-20	100	Lee et al., 1975
Cotton gin (no controls)	0.7	1-5	7	Lee et al., 1975
		<1	93	

*Source: Newman et al., 1981

NA = Not available

TABLE 2-3

Factors Affecting Air Residence Times for Sb-Containing Particulates

Factor	Reported Value	Reference
Dry deposition velocities	typically <0.5 cm/sec	Gorham, 1976
Enrichment factor (EF)[a]		
1) of Sb-enriched flue fly ash relative to crustal abundance	~100-1000 times	Newman et al., 1981
2) of Sb-enriched flue fly ash relative to concentration[a] in coal (Al reference)	3-7 (all in-stack particulates) 20-30 (<0.35 nm particulates)	Gladney et al., 1976
Washout factor (W)[b]	660	Bogen, 1974

[a] $EF = ([Sb]_S/[Al]_S)/([Sb]_C/[Al]_C)$
Where $[Sb\ or\ Al]_S$ and $[Sb\ or\ Al]_C$ represent the concentrations of antimony or aluminum in the particulate sample and the coal, respectively (Newman et al., 1981).

[b] $W = [Sb]_R/[Sb]_A$
Where $[Sb]_R$ is Sb concentration in rain, and $[Sb]_A$ is Sb concentration in air (Newman et al., 1981).

values from the literature (Newman et al., 1981). As mentioned above, the dry deposition velocity for Sb particulates is very low (<0.5 cm/sec). The rate of removal of Sb particulates by washout with rain is also low (W=660) compared to other particulates (Sc, Mn, Fe, Na and Cl, W=2000-13,000). This reflects not only the smaller particle sizes of the Sb particulates, but also the very low water solubility of the antimony species. Table 2-4 lists several estimations of the air residence times of Sb particulates. Estimated "air residence times" refers to the length of time it would take for the Sb particulates to be removed from the atmospheric compartment (taking into account particle size, absorption behavior, washout factors and other removal processes). The estimated residence times range from <30 days to ~40 days, which are much longer than the atmospheric residence times for most particulates (<1 week) (Duce et al., 1975). These longer residence times for Sb particulates are most likely related to the above mentioned small particle size, low washout coefficients and low dry deposition velocities.

Long air residence times indicate a potential for long-range transport. It is possible, therefore, that this occurs in the case of antimony particulates as their residence times appear to be sufficiently longer than most particulates (Newman et al., 1981). Only limited information is available on the presence of antimony oxides in the air. Sb_2O_3 has been detected in high concentrations (>300 ppm) downwind from a copper smelting plant (Crecelius et al., 1975). Antimony has been measured in urban areas at levels many times that predicted from the amount of antimony in the Earth's crust (Paciga and Jervis, 1976; Gladney et al., 1974). The incineration of products containing antimony substances and the combustion of fossil fuels are added sources of antimony in the atmosphere. These sources result in

TABLE 2-4

Estimated Air Residence Times for Sb Particulates

Material	Size Range (median size)	Residence Time	Reference
Most heavy metals (includes Sb)	5-20 nm	<30 days	Bowen, 1975
Sb-associated aerosols from air over Toronto	(1.2 nm)	somewhat longer than 30 days	Paciga and Jervis, 1976; Basu et al., 1981
Sb-associated aerosols from air over Boston	52-62%:0.4-0.75 nm 22-15%:0.75-1.5 nm rest:>1.5 nm	somewhat longer than 30 days	Gladney et al., 1974; Basu et al., 1981
Sb-containing particulates	NR	~40 days	Bowen, 1975

NR = Not reported

atmospheric concentrations of only 1-10 ng/m³ antimony substances (Gladney and Gordon, 1978; Greenberg et al., 1978). These levels are more than a million times lower than the lowest concentration to produce adverse effects in laboratory animals. Table 2-5 lists atsompheric antimony concentrations available in the literature (Newman et al., 1981). Sb concentrations for nonindustrial locations range from 0.05 ng/m³ (pristine, background site) to 88 ng/m³ (urban site). Higher concentrations of Sb particulates are found at industrial settings, with a high of 1210 ng/m³ being reported near smelting operations. These data support the conclusion that atmospheric Sb substances are mainly the result of local releases, not long-range transport. The Sb substances can be expected to accumulate in soils and in aquatic sediments, mainly surrounding producing and processing facilities (Federal Register, 1983a) (Sections 2.2. and 2.3.).

2.2. WATER

Markam et al. (1958) studied the photochemical properties of Sb_2O_3. Suspensions of prismatic Sb_2O_3 in both oxygenated and deoxygenated water darken when exposed to UV light. Suspensions of rhombic Sb_2O_3 darken only when exposed to UV light in deoxygenated water, not in oxygenated water. Sb_2O_3 absorbs strongly at wavelengths <325 nm; therefore, it is possible that suspensions of prismatic Sb_2O_3 will darken when exposed to sunlight at sea level. The proposed reaction pathway for photolysis of rhombic Sb_2O_3 in the absence of organic matter leads first to production of reduced antimony metal (dark, Sb°) and Sb_2O_5. In the presence of both oxygen and water, the Sb° is oxidized back to Sb_2O_3 (and, hence, no overall sign of darkening). The authors stated that the darkening of prismatic Sb_2O_3 even in the presence of oxygen was due to the prismatic crystal structure of Sb_2O_3 being resistant to rapid reoxidation with

TABLE 2-5

Atmospheric Antimony Concentrations[a]

Location	Setting	Date	Concentration (ng/m^3)	EF[b]	Reference
Over Atlantic Ocean (North 30°N)	background	1970-1972	0.05-0.64	2,300	Duce et al., 1975
Over Arabian Ocean	background	1973	0.05-0.64	NR	Sadasivan, 1978
Lake Windermere, England	rural	1970-1971	2.0	NR	Cawse and Peirson, 1972
Canada	rural	1971	0.13-0.60	NR	Cawse and Peirson, 1972
United Kingdom	rural/ suburban	1972	0.56-5.5	NR	Cawse and Peirson, 1972
New Jersey (USA)	rural	1976	<10	NR	Leaderer et al., 1978
Boston, MA (USA)	suburban	1970	1.3	500	Gladney et al., 1974
Boston, MA (USA	urban	1970	7.4	1,800	Gladney et al., 1974
Boston, MA (USA)	urban	1970	12.0	2,450	Gladney et al., 1974
Toronto (Canada)	urban	1974	8 (0.9-36)	1,270	Paciga and Jervis, 1976
Toronto (Canada)	urban	1974	6 (0.6-24)	1,000	Paciga and Jervis, 1976
Washington, DC (USA)	urban	1974	9.8 (1.8-26)	1,950	Kowalczyk et al., 1978
Heidelberg, Germany	urban	1971	5.1	NR	Bogen, 1974
Tampa, FL (USA)	urban	1975	0.40[c]	NR	Braman and Tompkins, 1978
USA	urban	1966	1.0	NR	Cawse and Peirson, 1972

TABLE 2-5 (cont.)

Location	Setting	Date	Concentration (ng/m³)	EF[b]	Reference
San Francisco, CA (USA)	urban	1970	1.1	NR	Bogen, 1974
United Kingdom	urban	1971	1.9	NR	Bogen, 1974
Niles, MI (USA)	urban	1969	5.8	NR	Bogen, 1974
New York, NY USA)	urban	1976	1-25	NR	Leaderer et al., 1978
East Chicago, IN (USA)	urban	1969	25	NR	Bogen, 1974
Beer Sheva, Israel	urban	1974	25 (15 50)	NR	Shani and Cohen, 1977
Ghent, Belgium	urban	1972	1-88	NR	Demuynck et al., 1976
Paris, France	urban	1970	50.8	NR	Bogen, 1974
Toronto (Canada)	industrial(A)	1974	45 (5-170)	8,300	Paciga and Jervis, 1976
Toronto (Canada)	industrial(B)	1974	17 (2-70)	2,270	Paciga and Jervis, 1976
Japan	industrial	1970	13-63	NR	Bogen, 1974
Chicago, IL (USA)	industrial	1970	1.4-55	NR	Bogen, 1974
Kellogg, ID (USA)	mining, smelting	1972	146 (5.2-1210)	46,000	Ragaini and Ondov, 1977

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[a] Source: Newman et al., 1981

[b] EF = Enrichment factor ($[Sb]_s/[Al]_s/[Sb]_c/[Al]_c$), where s = sample and c = Earth's crust

[c] Probably not total [Sb] due to analytical technique

NR = Not reported

oxygen. This difference in photochemical behavior was also noted by Mouzin (1980), who reported that the photodegradation of ordinary Sb_2O_3 is due to the presence of the cubic form, even in very small amounts. The orthorhombic form is photostable. Pertinent data regarding the photolysis of Sb_2O_4 and Sb_2O_5 were not located in the available literature as cited in the Appendix.

Oxidation of Sb_2O_3 has not been observed under environmental conditions (Newman et al., 1981). Pertinent data regarding the oxidation of Sb_2O_4 were not located in the available literature as cited in the Appendix. These reactions would not be expected to occur under environmental conditions.

Sb_2O_3 has a solubility of 2.9×10^{-5} g-atoms/ℓ in water at 25°C and pH range of 3-10 (Baes and Mesmer, 1976). Sb_2O_4 and Sb_2O_5 have been reported to be very slightly soluble in cold and hot water (Weast, 1973). Hydrolysis occurs under these conditions, with $Sb(OH)_3$ and $HSbO_2$ being the major species produced (Baes and Mesmer, 1976; Rai and Serne, 1978). These species exist in equilibrium with the solid Sb_2O_3, as well as with smaller amounts of $Sb(OH_2)^+$, SbO_2^- and SbO^+ (Rai and Serne, 1978). Figure 2-1 illustrates the various Sb solution species in equilibrium with Sb_2O_3 (solid) as a function of pH. Sb_2O_4 and Sb_2O_5 are much more resistant to hydrolysis than is Sb_2O_3. This is illustrated in Figure 2-2 (which represents a highly oxidizing environment). Figure 2-2 shows that all the oxide species are increasingly resistant to hydrolysis as the pH of the solution is raised. Table 2-6 lists selected pertinent equilibrium constants for reactions involving antimony oxides and their hydrolysis products. The very low solubility of the antimony oxides in water will severely limit the concentrations of the above mentioned antimony species.

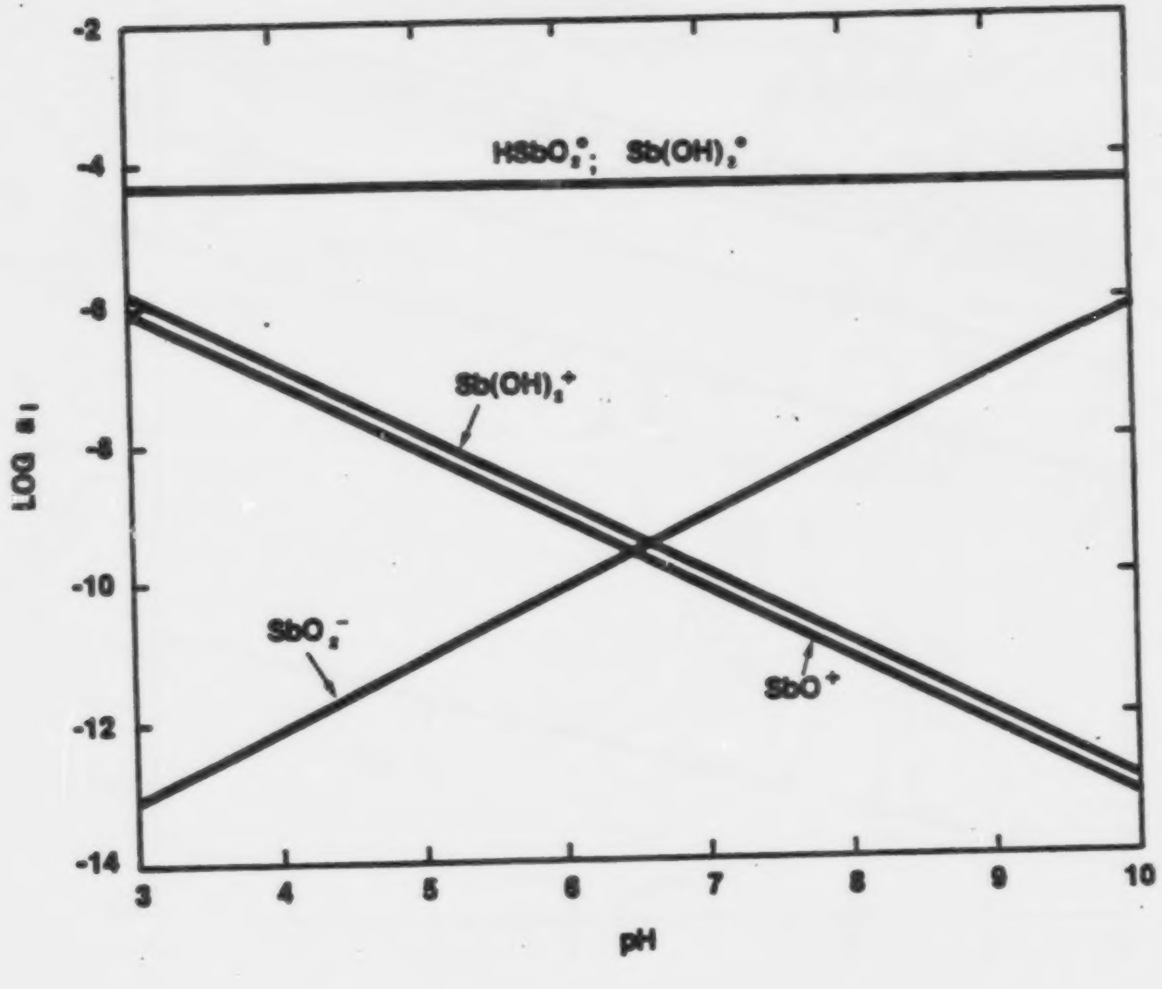

FIGURE 2-1

The activity of various antimony species in equilibrium with Sb_2O_3(s) (cubic). Oxidation-reduction changes do not affect the concentration of species reported in this figure (a_1 = activity coefficient of species 1).

Source: Rai and Serne, 1978

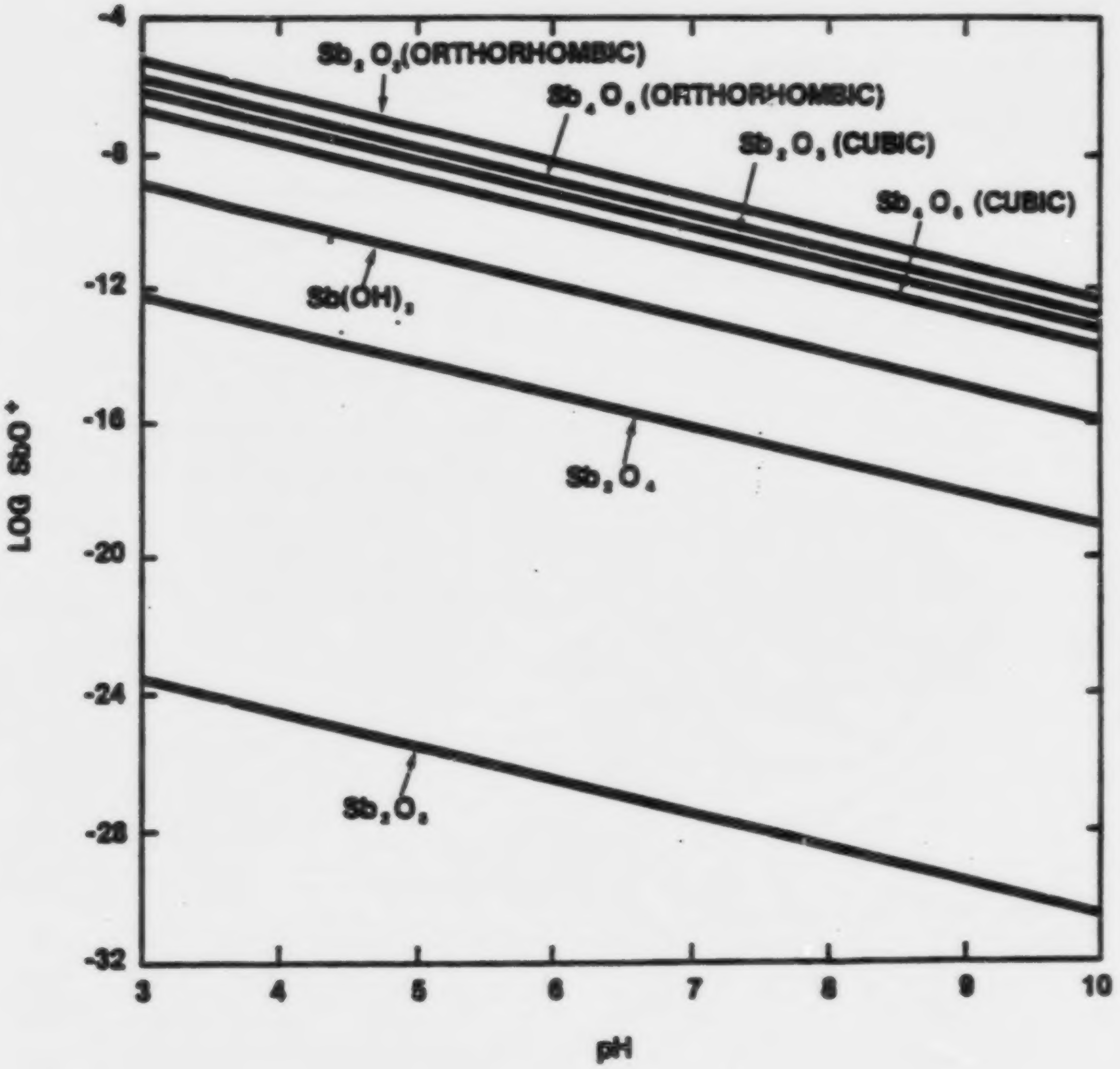

FIGURE 2-2

The Activity of SbO^+ in Equilibrium with Various Antimony Species in an Oxidizing Soil Solution Environment [$pO_2(g)$ = 0.68 atm]

Source: Rai and Serne, 1978

TABLE 2-6

Selected Equilibrium Constants (K) at 25°C and 1 atm for Reactions Involving Antimony*

Reaction	Log K
	Solid Compounds
Sb_2O_3 (cubic) + $2H^+ \leftrightarrow 2SbO^+ = H_2O$	-3.05
Sb_2O_3 (orthorhombic) + $SH^+ \leftrightarrow 2SbO^+ + H_2O$	-2.32
$Sb_2O_4 + 2H^+ \leftrightarrow 2SbO^+ + 0.5O_2 + H_2O$	-18.56
$Sb_4O_6 + 4H^+ \leftrightarrow 4SbO^+ + 2H_2O$	-14.96
$Sb(OH)_3 + H^+ \leftrightarrow SbO^+ + 2H_2O$	-5.93
$Sb_2O_5 + 2H^+ \leftrightarrow 2SbO^+ + H_2O + O_2$	-41.66
	Redox Reactions
$Sb(s) + H_2O \leftrightarrow SbO^+ + 2H^+ + 3e^-$	-10.53
$SbO^+ + H_2O \leftrightarrow HSbO_2^0 + H^+$	-1.19
$HSbO_2^0 \leftrightarrow SbO_2^- + H^+$	-11.79
	Hydrolysis and Other Reactions
$SbO^+ + H_2O \leftrightarrow Sb(OH)_3^0 + H^+$	-1.18
$SbO^+ + H_2O \leftrightarrow Sb(OH)^{2+}$	0.20
$SbO^+ + H_2O + NH_4^+ \leftrightarrow NH_4SbO_2^0 + 2H^+$	-12.98
$SbO^+ + F^- \leftrightarrow SbOF^\circ$	5.51

*Source: Rai and Serne, 1978

Pertinent data regarding the biological transformation of antimony oxides directly occurring in the environment were not located in the available literature as cited in the Appendix. A pure culture study using Stibiobacter senarmontii, an autotrophic bacterium isolated from Sb ore samples, demonstrated that biological transformation of antimony oxides in the environment could be possible (Lyalikova, 1974). The bacteria were grown in a mineral medium containing Sb_2O_3 and oxidized the chemical (Sb_2O_3) at rates of 45.5-51.6 mg/month for senarmonite (cubic) and 13.5-19.3 mg/month for valentinite (rhombic). Little Sb_2O_3 oxidation occurred in the sterile medium. Sb_2O_5 was the oxidation product, and Sb_2O_4 was also detected and was probably an intermediate.

Pertinent data regarding transformations of antimony oxides in sediments were not located in the available literature as cited in the Appendix.

Loss of antimony oxides from water through volatilization is very unlikely under normal environmental conditions due to their very low concentrations and the low concentrations of their hydrolysis products, and also due to their polarity and extremely low vapor pressures.

Most of the antimony oxides in water systems are present in suspended solid fractions (Shuman et al., 1977), not in the dissolved fraction as discussed above. The major transport modes for these oxides, as well as for other metals in aquatic systems, are as exchangeable species sorbed onto colloidal surface-active sites; as nonexchangeable Sb in organic matter; in association (i.e., coprecipitation or precipitation) with Al, Mn or Fe oxide coatings; and as part of the crystal lattices of silt, clay and silt minerals (Gibbs, 1973; Haynie, 1974; Shuman et al., 1977; Newman et al., 1981).

Shuman et al. (1977) studied polluted vs. nonpolluted sections of the Haw River, NC, with respect to the above modes of transport. Table 2-7 lists the results according to mode. It was found that 64% of the Sb in the polluted area was associated with Al, Mn and Fe oxides; the remainder was mainly found adsorbed by organic matter and in the crystalline state. At the unpolluted station, 95% of the Sb was found in the crystalline state. The overall suspended particulate Sb concentrations also differed for the two types of sites, with the polluted averaging 101 mg/kg (dry weight), and the control unpolluted stations averaging 12.5 mg/kg.

Apparently, the sorption and precipitation of Sb substances vary considerably depending upon the body of water studied. Crecelius et al. (1975) reported that the substantial quantities of antimony found in the sediments of Puget Sound were associated with precipitated hydrous iron and aluminum oxides, but found little Sb that was associated with organic matter. A study of the sediments of the North Adriatic Sea, however, found that the Sb present was associated primarily with organic matter, particularly humic acids (Strohal et al., 1975).

Antimony species appear to be removed from water primarily by coprecipitation with dissolved or colloidal organic and inorganic substances, or by settling of suspended particulates that adsorb antimony (U.S. EPA, 1979a). Increases in salinity (e.g., where river freshwater runs into seawater), changes in pH and slow current or stagnant conditions aid the precipitation and settling of both organic and inorganic substances (Sholkovitz, 1976; Stumm and Morgan, 1970; Crecelius, 1975).

TABLE 2-7

Percent Antimony Transported by Mode at Unpolluted and Polluted Stations*

Mode	Unpolluted	Polluted
Dissolved fraction		
Soluble	0.0	3.7
Suspended fraction		
Adsorbed	0.0	2.6
Oxide coating	1.6	64.0
Organics	3.3	10.3
Crystalline	95.2	12.6

*Source: Shuman et al., 1977

A study of Lake Washington, Seattle, WA, was done by Crecelius (1975) to determine the amounts and locations of the antimony substances present. As listed in Table 2-8, very little Sb is present in the dissolved state, the majority is present as suspended solids near the bottom and at or near the surface of the sediments.

Tables 2-9 and 2-10 list the results of studies of Sb content of the various water systems found in the literature (Newman et al., 1981).

Limited data are available regarding the bioconcentration of antimony oxides or antimony substances in general. It has been reported that Sb metal is bioconcentrated, although this appears to be quite variable (Basu et al., 1981). It has also been reported that the potential for accumulation is positive and that Sb can be concentrated 300 times by marine life (U.S. EPA, 1984a). BCFs for freshwater and marine invertebrates are reported to be 16,000 and for fish only 40. It has also been suggested, however, that BCFs of 1-100 for freshwater fish and benthic invertebrates indicate that bioconcentration is sufficiently low and that no further aquatic toxicity testing is necessary (Hildebrand, 1976; Shuman et al., 1977; U.S. EPA, 1978b; Federal Register, 1983a; Beck et al., 1981). Based on these BCFs regarding Sb for fish and the lower value for benthic invertebrates, bioconcentration is probably not significant in these organisms. The higher figure of 16,000 for freshwater and marine invertebrates reported above, however, indicates that bioconcentration of Sb may be significant in these organisms. Biomagnification of Sb, the indirect concentration of a compound as a result of the consumption of lower organisms, was not demonstrated to occur (Basu et al., 1981).

TABLE 2-8

Aquatic Antimony Content of Lake Washington, Seattle, WA*

Location	Concentration (μg/ℓ)
Dissolved	0.20
Suspended solids - near surface	2-6
near bottom	10-20
Sediments - surface (contaminated)	3-10
deep (uncontaminated)	0.4-0.8

*Source: Crecelius, 1975

TABLE 2-9

Aquatic Antimony Concentrations*

Water type	Location	Date	Concentration (µg/ℓ)	Comment	Reference
Rivers					
Mississippi	Minneapolis, MN (USA)	1965	0.32	dissolved	Kharkar et al., 1968
Susquehanna	Marietta, PA (USA)	1966	0.98	dissolved	Kharkar et al., 1968
Mad	Blue Lake, CA (USA)	1966	0.55	dissolved	Kharkar et al., 1968
Klamath	Lamath Glenn, CA (USA)	1966	0.47	dissolved	Kharkar et al., 1968
Russian	Calif. Hwy. 116, CA (USA)	1966	0.67	dissolved	Kharkar et al., 1968
Eel	U.S. Hwy. 101, CA (USA)	1966	0.27	dissolved	Kharkar et al., 1968
Brazos	U.S. Hwy. 59, TX (USA)	1966	0.34	dissolved	Kharkar et al., 1968
Rhone	Avignon, France	1966	1.53	dissolved	Kharkar et al., 1968
Amazon	Santarem, Brazil	1965	4.90	dissolved	Kharkar et al., 1968
Haw	North Carolina (USA)	1973-1974	3-224	total	Haynie, 1974
Hillsborough	Tampa, FL (USA)	1975	0.05	dissolved	Tompkins, 1977
Lakes					
Washington	Seattle, WA (USA)	1975	0.20	dissolved	Crecelius, 1975
Carroll	Tampa, FL (USA)	1975	0.05	dissolved	Tompkins, 1977
Marine					
Adriatic Sea	Rovinj, Yugoslavia	1974	0.17-0.53	dissolved	
			27.0-91.0	total	Strohal et al., 1975
Irish Sea	Scotland	1965	0.13-0.40	dissolved	Portmann and Riley, 1966
Hillsborough Bay	Tampa, FL (USA)	1975	0.02-0.03	dissolved	Tompkins, 1977
Rainfall					
USA	Seattle, WA	1975	3.5	total	Crecelius, 1975
Germany	Heidelberg, Germany	1971	2.6	total	Bogen, 1974
USA	Quillayute, WA	1970	0.18	dissolved	Rancitelli and Perkins, 1970
United Kingdom	Wraymires	1973	1.8	total	Peirson et al., 1973
Potable					
USA	194 public water supplies	1962	25.0 (1-1000)	finished	Taylor, 1963
USA	94 municipal water supplies	1966	0.2	tap	Schroeder, 1966

*Source: Newman et al., 1981

TABLE 2-10

Antimony Content of Sediments and Suspended Solids*

Sample	Location	Date	Concentration mg/kg (dry wt)	Comment	Reference
Sediments--marine and estuarine					
N. Adriatic Sea	Rovinj, Yugoslavia	1974	20	basin received industrial effluents	Strohal et al., 1975
Puget Sound	Seattle, WA (USA)	1970-1972	0.3-1.0	uncontaminated sediments	Crecelius et al., 1975
Puget Sound	Seattle, WA (USA)	1970-1972	up to 10,000	contaminated surface sediments	Crecelius et al., 1975
Long Island Sound	New York (USA)	1972	0.3-11.0	160 sampling sites	Greig et al., 1977
Saronikos Gulf	Athens, Greece	1973	0.3-0.7	background	Griggs et al., 1978
Saronikos Gulf	Athens, Greece	1973	2-75	contaminated	Griggs et al., 1978
Sediments--freshwater					
Lake Washington	Seattle, WA (USA)	1975	3-10	surface (contaminated) sediments	Crecelius, 1975
Lake Washington	Seattle, WA (USA)	1975	0.4-0.8	deep (uncontaminated) sediments	Crecelius, 1975
Haw River	North Carolina (USA)	1973-1974	0.1-4.0	upstream of cities	Haynie, 1974
Haw River	North Carolina (USA)	1973-1974	0.1-29.2	downstream of cities	Haynie, 1974
Sediments--freshwater					
Lake Huron	(USA)	1974-1975	0.8-1.3	surface (0-3 cm)	Robbins, 1980
Lake Huron	(USA)	1974-1975	0.2-0.5	deep (10-50 cm)	Robbins, 1980
Lake Erie	(USA)	1972	2-6	surface (0-30 cm)	Walters et al., 1974
Lake Erie	(USA)	1972	0.2-1.0	deep (30-250 cm)	Walters et al., 1974
Danube River	Vienna, Austria	1973-1974	8-22		Rehwoldt et al., 1975
Woodhull Lake	Adirondack Mts., NY (USA)	1975	0.1-0.2	deep (20-40 cm)	Galloway and Likens, 1977
Woodhull Lake	Adirondack Mts., NY ([illegible]	1975	1.2-3.0	surface (0-20 cm)	Galloway and Likens, 1977
Coeur d'Alene River	Idaho (USA)	1971	270-900	contaminated (mining)	Maxfield et al., 1974

TABLE 2-10 (cont.)

Sample	Location	Date	Concentration mg/kg (dry wt)	Comment	Reference
Suspended solids					
Lake Washington	Seattle, WA (USA)	1975	2-6	near surface	Crecelius, 1975
Lake Washington	Seattle, WA (USA)	1975	10-20	near bottom	Crecelius, 1975
Two rivers into Lake Washington	Washington (USA)	1975	1.6	composite	Crecelius, 1975
Eight rivers into Puget Sound	Washington (USA)	1972-1973	0.2-4.2	composite	Crecelius et al., 1975
Haw River	North Carolina	1973-1974	7-16	upstream of effluents	Haynie, 1974
Haw River	North Carolina	1973-1974	79-123	downstream of effluents	Haynie, 1974
Danube River	Vienna, Austria	1973-1974	2.9-5.2		Rehwoldt et al., 1975

*Source: Newman et al., 1981

In summary, some photochemical reduction to metallic Sb and Sb_2O_5 of both suspended prismatic and rhombic Sb_2O_3 occurs upon irradiation at wavelengths >290 nm. The Sb metal from the rhombic form is readily oxidized back to Sb_2O_3 in oxygenated waters, while the prismatic form darkens due to the absence of facile oxidation of the dark Sb metal in the crystal lattice. It appears that very little of the Sb oxides in water occur in the dissolved state, and that which does dissolve is present as various hydrolysis products. The vast majority of Sb species in water systems are in the form of various suspended particulates that tend to settle onto sediments over time. The rate of removal from water by precipitation or by precipitation and settling depends on such factors as salinity, changes in pH and amount of current or turbulance present in the water systems. Sb associated with particulates would therefore be expected to accumulate, for example, where contaminated rivers flow into reservoirs, lakes, delta areas and other areas where sediments are actively being deposited, such as inside of bends in rivers (Newman et al., 1981). The potential for this accumulation increases as the source(s) of the Sb material is approached. Very little, if any, loss of the antimony oxide is expected through volatilization. Bioconcentration of antimony species other than metallic Sb has been shown to be insignificant for most aquatic species; however, a report of a BCF of 16,000 for freshwater and marine invertebrates indicates that bioconcentration may be significant for some species; biomagnification of Sb materials was not observed.

2.3. SOIL

Pertinent data regarding the chemical transformations of Sb_2O_4 and SB_2O_5 in soil were not located in the available literature as cited in the Appendix. It has been reported that Sb_2O_3 may react with bases in alkaline soils, antimonates being the products formed (U.S. EPA, 1976).

Pertinent data regarding the hydrolysis of antimony oxides were not located in the available literature as cited in the Appendix. Based upon the very low water solubility of these oxides (see Section 2.2.), significant hydrolysis is not expected.

Pertinent data regarding the photolysis of antimony oxides were not located in the available literature as cited in the Appendix. Based upon the photolytic behavior of these oxides in suspension in water (see Section 1.2.), photolysis is not expected to be very significant, and would occur only at the very surface of the soil. Pertinent data regarding the biodegradation of antimony oxides were not located in the available literature as cited in the Appendix. Based on the fact that an autotrophic bacterium, S. senarmontii, has been found in Sb samples, which has been shown in pure culture studies to derive energy from Sb_2O_3, biotransformations of antimony oxides in the soil may be possible (Lyalikova, 1974) (see Section 1.2.).

Loss of antimony oxides due to volatilization from soil is not expected due to the very low vapor pressures of the oxides under environmental conditions.

Trace metals, including Sb, have been reported to be found in soils as water soluble species in the interstitial water, precipitates and coprecipitates as oxides, exchangeable species absorbed onto soil surfaces and organically bound species, and also within mineral crystal lattices (Newman et al., 1981). As mentioned above, the amount of water soluble species is not expected to be significant for the antimony oxides. Precipitation and/or coprecipitation as oxides (Sb_2O_3, Sb_4O_6 and Sb_2O_4) has been suggested as important porcesses by which Sb is retained on soil surfaces (Newman et al., 1981). Pertinent data regarding the leaching of antimony oxides were not located in the available literature cited in the Appendix.

An abstract for a study published by Refel et al. (1983) was located which concerned soil migration of Sb_2O_5 and other Sb substances. Improper landfilling and disposal of slag from antimony oxide production, combustion and use facilities, and sludge from municipal sewage treatment plants and solid wastes from municipal incinerators may result in release of antimony to the aquatic environment, including groundwater and surface water supplies (Basu et al., 1981).

Pertinent data regarding the presence of antimony oxides in groundwater were not located in the available literature as cited in the Appendix. Based on the very low solubility of these compounds (see Section 1.2.), significant levels would not be expected in the groundwater except perhaps in the immediate vicinity of plants that manufacture or use antimony substances, or disposal sites. Taylor (1963) surveyed 194 public finished water supplies in the United States and found a range of Sb concentrations of 1-100 μg/ℓ, with a reported mean value of 25 μg/ℓ. Schroeder (1966) surveyed 94 municipal water supplies, however, and reported values generally <0.2 μg/ℓ in tap water. It was also mentioned that tap water concentrations of Sb may increase with increased softness of the water due to leaching of Sb from plumbing (Schroeder and Kraemer, 1974). It would appear from these data that at least the municipal water systems tested have not been contaminated significantly by antimony oxides. More current data regarding Sb in municipal water systems as well as groundwater aquafers would be useful.

In summary, antimony trioxide (and presumably Sb_2O_4 and Sb_2O_5) is apparently persistent in soil (Crecelius et al., 1975), which is to be expected from its low water solubility, lack of reactivity, stability and low vapor pressure. It is expected that antimony substances will accumulate in the soil and sediment near production and processing facilities (Federal Register, 1983a), as well as at or near disposal sites. Table 2-11 supports this conclusion with data indicating that the Sb content of soils is much greater in contaminated sites near smelters and mines than in soil from background sites (Newman et al., 1981).

TABLE 2-11

Antimony Content of Soils*

Sample (cm)	Location	Date	Concentration mg/kg (dry wt)	Comment	Reference
0-2	Kellogg, ID (USA)	1972	18-20	background sites	Ragaini et al., 1977
0-2	Kellogg, ID (USA)	1972	104-260	contaminated sites (smelter)	Ragaini et al., 1977
0-3	Tacoma, WA (USA)	1972	3-5	background sites	Crecelius et al., 1974
0-3	Tacoma, WA (USA)	1972	8-204	contaminated sites (smelter)	Crecelius et al., 1974
0-5	Dubrave, Czechoslovakia	1976	490-8500	contaminated site (mine tailings)	Banasova, 1976

*Source: Newman et al., 1981

3. EXPOSURE

3.1. AIR

Most of the available data concerning exposure to antimony substances do not distinguish between antimony metal, Sb_2O_3 and Sb_2S_3 (see Section 1.2.). It is expected, however, that Sb exists as the trioxide in the atmosphere (Basu et al., 1981). This conclusion is due mainly to the fact that most of the atmospheric releases of antimony substances result from high temperature industrial processes; the combustion of petroleum, petroleum products and coal; and the incineration of products that contain antimony. At the high temperatures used in these processes, oxidation of the antimony substances occurs, resulting in the formation of Sb_2O_3 (and possibly also Sb_2O_4 and Sb_2O_5) (Newman et al., 1981). The U.S. EPA (Federal Register, 1983a) reported that it expects a minor contribution to environmental levels of antimony substances from the mining and hauling of antimony-containing ores. Table 2-5 summarized the available data concerning the antimony content in total suspended particulate samples of the atmosphere in various environmental settings (Newman et al., 1981). The risk of human exposure depends upon proximity to industrial and municipal emitters. Sb concentrations may range over 6 orders of magnitude (0.05 ng/m^3-pristine, background site, to 1210 ng/m^3, near ore-smelting operations) depending on environmental setting (rural, urban, industrial). While concentrations of 300 ppm antimony have been reported 40 km downwind from industrial sources (Crecelius et al., 1974, 1975), ambient levels for urban and rural areas on the order of 10^{-7} to 10^{-5} ppm are suggested by the available monitoring information (Basu et al., 1981).

The antimony released from high temperature processes condenses rapidly onto suspended particulate matter (Basu et al., 1981) (see Section 2.1.).

The emitted antimony will associate predominantly with small diameter particles (e.g., 1 nm in size) (Gladney et al., 1974; Paciga and Jervis, 1976), which are not only difficult to trap with conventional stack technology, but also tend to settle out less rapidly and thus are transported greater distances through the atmosphere (Basu et al., 1981). Due to their small diameter, these particulates may also penetrate deeply into the pulmonary tract of man. Therefore, the greatest risk of general population exposure to antimony substances by inhalation is due to the presence of antimony-containing particulates in ambient air. As noted above, however, the ambient levels reported for both urban and rural air are very low. Therefore, it appears from monitoring data that human exposure to antimony through inhalation will be significant only in the immediate vicinity of, or directly downwind from industrial and municipal dischargers.

Little information was found regarding the human exposure associated with proximity to any one of these sources of antimony. Jervis et al. (1977) and Chattopadhyay et al. (1977) examined the use of scalp hair as a monitor of human exposure to environmental pollutants (including antimony) as a function of proximity to three secondary lead refineries in central Toronto. The hair of exposed population groups (those living near refineries) has ~1.5 times the antimony content of urban residents (average of 14.6 vs. 9.7 ppm, respectively) and nearly 2 times that of rural residents (7.9 ppm).

3.2. WATER

Antimony and its compounds are released to water from many different sources (Basu et al., 1981). Effluent and slag from mining, processing and smelting of ores, coal-fired power plants and municipal incinerators; sludge from sewage treatment plants; and spent catalysts are all major sources of release into water systems. Another source of antimony substances in water is the settling and washout of Sb-containing particulates from the atmosphere. This is expected to be significant only near production and processing facilities.

Data regarding levels of release of antimony substances to water systems included a report that a copper smelter in Tacoma, WA, was found to emit $2x10^3$ kg antimony/year in effluents that were discharged directly into Puget Sound (U.S. EPA, 1976). Additionally, $1.5x10^6$ kg antimony/year was dumped directly into the Sound in the form of crystalline slag particles. The industrial polymerization of dimethyl terephthalate and ethylene glycol results in the production of $9.82x10^4$ to $1.96x10^5$ kg of waste antimony/year (NIOSH, 1977). Improper landfilling of this and other waste material may result in release to and subsequent contamination of groundwater and surface water supplies.

Available data regarding dissolved aquatic antimony concentrations at various sites were listed in Table 2-9, and Table 2-10 listed antimony content of sediments and suspended solids (Newman et al., 1981). The largest amount of dissolved antimony reported was 4.90 μg/ℓ in water from the Amazon River in Brazil. Most of the other data were generally <1 μg/ℓ dissolved Sb. Since most of the metal (and its compound) is associated with crystalline minerals and the solubilities of the substances are very low, the majority of the antimony present is expected to be in the form of suspended or precipitated particles in the water and sediments. The data in Table 2-9 concerning total vs. dissolved solids and the data in Table 2-10 confirm this prediction.

While antimony has been found in waters throughout the United States, the levels found may not be indicative of the potential for human exposure from contaminated drinking water supplies. Concentrations ranging from 0.016 ppm Sb in Cincinnati drinking water to 0.057 ppm in Atlanta were reported by Blanchard et al. (1959). A later survey by Schroeder (1966) of 94 municipal water supplies reported that levels were on the average <0.2 ppb when measured in tap water. Schroeder and Kraemer (1974), however, noted that Sb levels can be increased in soft water supplies due to the leaching of Sb from plumbing. The ambient water quality criterion for Sb has been established as 145 μg/ℓ (0.145 ppm) (U.S. EPA, 1980). Drinking water was assumed to contribute 99% of the assumed exposure, while eating contaminated fish accounted for 1%.

Pertinent data regarding contamination levels of groundwater were not located in the available literature as cited in the Appendix.

3.3. FOOD

Tanner and Friedman (1977) reported the results of an FDA survey of various trace metals including antimony in various food classes. Table 3-1 lists the results, which range from a low median level of <0.004 ppm (wet weight) in dairy products to a high of <0.01 ppm in grain and cereal products. Based on these results, the daily intake for antimony was calculated to be too negligible to assign a meaningful value.

Earlier reports indicated significant amounts of dietary intake of antimony assimilated daily. These reports range from an average dietary intake for man of <100 μg/day (Schroeder, 1970) to 0.25-1.28 mg/day for institutionalized children (Murthy et al., 1971). Differences in analytical methodology were suggested as contributing factors in the discrepancy between these two reports and the FDA survey (Tanner and Friedman, 1977) cited above (U.S. EPA, 1980). The very low antimony levels of dietary classes in the United States was supported by Clemente (1976), who reported a mean value of several micrograms Sb daily intake in Italian diets.

TABLE 3-1

FDA Survey of Antimony in Various Food Classes*

Food Class	(ppm, wet weight)	
	Median Level	Range
Dairy products	<0.004	<0.002-0.002
Meat, fish and poultry	0.008	<0.004-0.015
Grain and cereal porducts	<0.01	0.006-0.05
Leafy vegetables	<0.006	0.001-0.027
Legume vegetables	0.008	<0.002-0.014
Garden fruits	<0.006	0.002-0.011

*Source: Adapted from Tanner and Friedman, 1977

Sb_2O_3 can be concentrated 300 times by marine life (U.S. EPA, 1984a) (see Section 2.2.). The Sb concentration factors were reported to be 16,000 for freshwater and marine invertebrates and 40 for fish. The U.S. EPA (1983) reported BCFs of 1-100 for freshwater fish and benthic invertebrates. Because no other information was available, the U.S. EPA (1980) suggested a weighted average of 1 for antimony and the edible portion of all freshwater and estuarian aquatic organisms consumed by Americans.

3.4. DERMAL

Sb_2O_3 is used in many consumer products as a pigment and/or flame retardant. The potential for consumer exposure to Sb_2O_3 from its use in plastics and textiles is quite low (Basu et al., 1981). Exposure from paints is possible because of paint chalking. It has been reported, however, that Sb_2O_3 is less chalky than other white pigments used in paints (U.S. EPA, 1976). Potential leaching of Sb_2O_3, as well as other metallic components, from ceramic glazes and porcelain enamels has led to guidelines to limit the levels of these materials used in pottery ware (Basu et al., 1981).

3.5. TOTAL EXPOSURE ESTIMATES

The total estimated daily intake of antimony by human populations in the United States is believed to be quite small and even negligible relative to other environmental agents of concern (e.g., cadmium, lead or mercury) (U.S. EPA, 1980). Based on the most recently available data concerning dietary antimony intake (Tanner and Friedman, 1977), no appreciable additional amounts of antimony in the diet would be expected (U.S. EPA, 1980b). This also applies to the amounts of antimony being ingested by water consumption.

An individual would be exposed to a total of only 1.7 μg/day Sb in the highest recorded ambient air level (0.085 μg/m^3) for an urban setting, assuming a daily inhalation rate of 20 m^3. Clemente (1976) concluded that the daily intakes of selected Italian populations were <2.0 μg/day. This is considerably lower than the ADI for man calculated by the U.S. EPA (1980).

4. PHARMACOKINETICS

4.1. ABSORPTION

Pertinent data regarding GI and respiratory system absorption of antimony oxides were not located in the available literature as cited in the Appendix. Absorption data concerning radioactively labeled related compounds have been published. Felicetti et al. (1974a) used oral and aerosol exposure in Syrian hamsters to ^{124}antimony tartrate in trivalent and pentavalent form to study absorption. These authors found absorption levels to be quite low, at levels of 1 or 2% of the ingested dose, and presented comprehensive retention and distribution data. Other experiments using ^{124}antimony tartrate have been done in the beagle dog, using aerosols and inhalation exposure (Felicetti et al., 1974b). Tissue retention data were also presented. Djuric et al. (1962) presented data on absorption, distribution and excretion in rats after inhalation of ^{124}antimony chloride; studies were also done with this compound in rabbits and dogs. Thomas et al. (1973) also used aerosols containing ^{124}antimony tartrate complexes, and exposed mice to particles formed at different temperatures. Retention and distribution data were presented in these studies. Elinder and Friberg (1977) found that 15% of tartar emetic (potassium antimony tartrate) is absorbed from the GI tract of mice.

Antimony absorption from the respiratory tract is a function of particle size and solubility in the lung, the latter being dependent upon the chemical form of the antimony compound (Djuric et al., 1962; Felicetti et al., 1974b; Thomas et al., 1973). In humans, Belyaeva (1967) found detectable levels of antimony in placental tissue, amniotic fluids and cord blood in pregnant women who worked in antimony smelters during pregnancy. Information concerning absorption of antimony through the skin is sparse. Gross et al. (1955a), using antimony trioxide dust dispersed into a paste (25 nm) and applied to rabbit skin, found no systemic or tissue effects.

4.2. DISTRIBUTION

Studies on the distribution of antimony in the tissues of rats after repeated oral administration of antimony trioxide or potassium antimony tartrate are summarized in Table 4-1. Although average daily doses and total cumulative doses varied by a factor of ~1000, variations of antimony levels within the specified organs varied by a factor of <8. While a relationship of tissue levels in liver, lung and spleen to total cumulative dose is apparent when comparing the two low-dose studies (Schroeder et al., 1970; Westrick, 1953) with the high-dose study by Sunagawa (1981), the overall pattern suggests relatively rapid tissue saturation, with increasing doses or durations of exposure causing only moderate increases in tissue levels of antimony. Based on the data presented in Table 4-1, both antimony trioxide and potassium antimony tartrate present similar patterns of tissue distribution.

Felicetti et al. (1974a) presented distribution data in hamsters using aerosols of radioactive antimony tartrate (NIOSH, 1978; U.S. EPA, 1980). Distribution can be influenced by the temperature at which antimony aerosols are generated (Thomas et al., 1973). These authors speculated that the antimony tartrate aerosol used in their experiments became an oxide at the two higher temperatures used. Intravenous administration of 1.3 mg antimony (tartar emetic)/kg to three monkeys led to peak blood antimony levels of 125-190 μg antimony/deciliter at 15 minutes, followed by a rapid decrease to 10-20 μg antimony/deciliter after 24 hours (Waitz et al., 1965).

TABLE 4-1

Distribution of Antimony in the Tissue of Rats After Repeated Oral Adminsitration of Antimony Trioxide and Potassium Antimony Tartrate

Chemical Form:	Potassium Antimony Tartrate	Antimony Trioxide	Antimony Trioxide	Antimony Trioxide	Antimony Trioxide
Vehicle:	Drinking water	Gelatin capsules	Food	Food	Food
Duration of Exposure:	~1000 days	20 days	168 days	168 days	243 days
Concentration:	13.7 mg/ℓ (5mg/ℓ as Sb)	NA	1%	2%	2%
Average Daily Dose:	1.07 mg/kg bw[a] (0.36 mg/kg as Sb)	13 mg/kg bw (5.4 mg/kg as Sb)	500 mg/kg bw[b] (209 mg/kg as Sb)	1000 mg/kg bw[b] (418 mg/kg as Sb)	1000 mg/kg bw (418 mg/kg as Sb)
Cumulative Dose (Sb):	360 mg/kg	108 mg/kg	35,112 mg/kg	70,224 mg/kg	101,574 mg/kg
Organ Levels (μg/g):					
Adrenal	67.0	NM	NM	NM	NM
Blood	NM	NM	148.11	114.87	NM
Bone	NM	NM	NM	NM	2.48
Heart	12.10	7.6	NM	NM	5.08
Kidney	10.14	6.7	NM	NM	6.02
Liver	11.57	8.9	15.19	17.74	15.50
Lung	17.67	14.0	28.84	27.26	3.70
Spleen	15.97	18.9	61.88	55.71	8.06
Thyroid	NM	88.9	NM	NM	156.25
Reference:	Schroeder et al., 1970	Westrick, 1953	Sunagawa, 1981	Sunagawa, 1981	Gross et al., 1955a

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[a] Assuming rats consumed the equivalent of 7.0% of their body weight in water/day.

[b] Assuming rats consumed the equivalent of 5% of their body weight in food/day.

NM - Not measured

NA - Not applicable

Djuric et al. (1962) carried out distribution and excretion experiments on rats who inhaled an aerosol of ^{124}antimony chloride. Two rabbits and one dog were also given intratracheal doses of the same compound. Inhalation exposure resulted in a rapid early loss from lung tissue, with distribution of the compound to whole blood, spleen, heart and other vascularized tissues. Localization of antimony in red cells of the rabbit and dog was not seen.

Blood is the main vehicle for transport of absorbed antimony to various tissues of the body (U.S. EPA, 1980). Relative partitioning of antimony between the red blood cells and plasma is a function of valency with trivalent antimony carried in red cells and pentavalent antimony carried in plasma. Levels attained and clearance depend on route of intake, chemical and physical form, and exposure parameters such as particle size. Distribution data using antimony tartrate have been presented by Thomas et al. (1973), Felicetti et al. (1974b) and Djuric et al. (1962). There are also differences in distribution depending on the species of animal studied. In rats, concentration of trivalent forms is greater in erythrocytes (Djuric et al., 1962). The rat also has the longest biological half-time (U.S. EPA, 1980).

In animals, trivalent antimony (tartrate) aerosols produce highest levels of radioactive tracer in lung, skeleton, liver, pelt and thyroid (Felicetti et al., 1974a,b; Thomas et al., 1973; U.S. EPA, 1980). Parenteral administration to animals produces accumulation of trivalent antimony (tartrate) in liver and kidney, as well as in pelt and thyroid (Molokhia and Smith, 1969; Waitz et al., 1965). Clearance from blood to tissues is rapid, especially with parenteral administration.

Casals (1972) used radioactive antimony dextran glycoside in mice, rats and rabbits and found that serum levels decreased rapidly. Normal levels of tissue distribution in rats for antimony were presented by Kostic et al. (1977). The U.S. EPA (1980) concluded that tissue distribution of inhaled antimony is a function of valency state with levels of trivalent antimony, increasing more rapidly in liver than the pentavalent form. Skeletal uptake is greater with pentavalent forms. Parenteral administration tends to show greater accumulation in kidneys, followed by liver and mineralized tissue (Molokhia and Smith, 1969; Waitz et al., 1965). In humans, antimony accumulates in selected soft tissues, especially the kidneys, liver, thyroid, certain endocrine organs and, to some extent, the heart.

Brune et al. (1980) studied distribution of trace elements in kidney, liver and lung from autopsy samples of workers retired from an antimony smelter and refinery. Median levels in lungs were significantly greater than controls; exposed individuals had median levels of 0.32 ppm and controls, 0.029 ppm.

Measurement and detection of antimony in process workers lungs _in vivo_ using x-ray spectrophotometry was performed by McCallum (1963, 1967) and McCallum et al. (1971). Abdalla and Saif (1962) demonstrated that in man, chemotherapeutic use of trivalent (organic) antimony for schistosomiasis leads to highest accumulation in liver, thyroid and heart.

4.3. METABOLISM

Most information regarding metabolism of antimony concerns organic derivatives (antimonials) and is of doubtful significance for trioxides and pentoxides. There is little storage of antimony. When storage does occur, it is located in liver, red blood cells, thyroid and parathyroid, but most distribution is nonspecific. Little else is known about metabolism

(Stokinger, 1981). Tissue distribution patterns are similar to distribution with organic antimony compounds. Inhalation exposure of rats to trivalent ^{124}antimony chloride resulted in atypical, rapid loss from the lung with a somewhat longer biological half-life (100 days) in the lung after the 20th day (Djuric et al., 1962). The in vivo work of Westrick (1953) with 2% antimony trioxide showed little effect on oxygen uptake or body weight in rats, however when thyroxin was given along with antimony trioxide, increased body weight loss occurred, with increased oxygen uptake.

4.4. EXCRETION

The only information available on the excretion of antimony oxides comes from the study by Gross et al. (1955a) in which rats were administered antimony trioxide. After single oral doses of 200 mg antimony trioxide in 5 mℓ of water, only 3.24% of the dose was eliminated in the urine. Levels in the feces were not measured. After administration of antimony trioxide in the diet at a concentration of 2% for 8 months, antimony excretion in the feces was much greater than in the urine. As summarized in Table 4-2, tissue levels of antimony dropped by ~40-90% in a group of rats held for 40 days after exposure had terminated, with the greatest reductions noted in the liver and GI tissue. It cannot be determined from the report if the slight increase in the levels of antimony in the muscle were significant. Relative excretion by urine or feces depends to a great extent on the nature of the antimony compound. Gellhorn et al. (1946) observed that hamsters injected intraperitoneally with four different preparations excreted 50% of the antimony of the trivalent compound in the feces, but the majority of the pentavalent compound was excreted by the kidneys. Goodwin and Page (1943) found that 30-40% of the antimony of pentavalent compounds was excreted in the urine during the first 1-2 hours, while tartar emetic (potassium

TABLE 4-2

Distribution of Antimony in Tissue of Rats Fed Antimony Trioxide*

Organ	Antimony (as Sb_2O_3) Immediately After End of Exposure (µg/g)	Antimony (as Sb_2O_3) 40 Days After End of Exposure (µg/g)	% Reduction
Thyroid	156.25	75.40	51.7
Spleen	8.06	4.75	41.1
Kidney	6.02	2.66	55.8
Heart	5.08	1.80	64.6
Bone	2.48	0.91	63.3
GI content	1059.70	0.81	~100
Muscle	0.30	0.55	-83.3
Lungs	3.70	1.20	67.6
Liver	15.50	9.24	90.3
GI tissue	10.74	1.50	86.0

*Source: Gross et al., 1955a

antimony tartrate) did not show such a high initial rate. Boyd and Roy (1929) found that 20% of the pentavalent compound was recovered from urine in the first 2 1/2 hours, while Hassan (1938) reported that trivalent antimony administered to patients was excreted to the extent of 8.6-10.8% within the first 24 hours.

Excretion data from studies using antimony tartrate were presented by Felicetti et al. (1974a) for the hamster and by Felicetti et al. (1974b) for the dog. Data for rats after inhalation of antimony chloride were presented by Djuric et al. (1962) and for mice with antimony tartrate by Thomas et al. (1973). Half-times for the slower component of elimination range from ~16-100 days.

After inhalation, excretion occurs by both GI and renal routes. Parenteral administration of trivalent antimonials causes rapid urinary excretion in guinea pigs, dogs and humans (Otto and Maren, 1950; Abdalla and Saif, 1962), while fecal clearance is more important with hamsters, mice and rats. Much of this information was gathered from studies using organic antimony compounds (NIOSH, 1978).

Clemente (1976) used neutron activation analysis to determine that <0.3 μg was excreted in a normal Italian population. Under conditions of occupational exposure, urinary excretion is elevated but highly variable from person to person (U.S. EPA, 1980).

5. EFFECTS

5.1. CARCINOGENICITY

Positive oncogenic effects were observed in 15/17 female rats exposed to air containing 4.2 mg/m^3 (0.22 mg/kg/day) antimony trioxide for 6 hours/day, 5 days/week for 1 year (Asarco, Inc., 1980; Watt, 1980, 1981, 1983). A statistically significant increase in the number of lung tumors in exposed rats was observed. Animals exposed to 1.6 mg/m^3 and control animals did not develop lung tumors. Watt (1983) further reports the results of studies exposing female CDF rats and S-1 miniature swine to antimony trioxide dust at levels close to the TLV. Rats and swine were exposed to 1.6 (±1.5) mg/m^3 (as Sb) or 4.2 (±3.2) mg/m^3 (as Sb) for 6 hours/day, 5 days/week for 1 year. Some of the rats were observed for another year after termination of exposure. Serum chemistry, enzyme levels and hematological parameters were evaluated. Gross and histological examination of tissues was also performed.

Body weights were significantly higher in exposed rats. Lung alterations were evident in exposed animals, including statistically significant increases in lung weight and a mottled appearance. No exposure related alterations were observed in swine.

In rats, primary lung neoplasms were observed, with the incidence of both neoplastic and non-neoplastic lesions increasing with exposure level and length of time of exposure. Most neoplasms occurred at the end of the exposure time in the high dose rats. Neoplasms were scirrhous carcinomas, squamous-cell carcinomas or bronchioalveolar adenomas. Non-neoplastic responses were observed in both high- and low-dose rats. Liver and mammary tumors were noted in both exposed and control groups. Other researchers have suggested a relationship between antimony exposure and pulmonary carcinogenesis. The time when tumors first appeared was not noted in the Asarco, Inc. (1980) rat study, so that duration of exposure and latency to tumor formation cannot be determined. Data are summarized in Table 5-1.

Antimony trioxide was selected for an NCI bioassay; however, the chronic feeding study has been halted pending evaluation of the subchronic study in which little toxicity was found (Watt, 1981). As a result, the bioassay results will not be available for several years. Wong et al. (1979) studied inhalation of antimony trioxide, antimony ore concentrate and thallic oxide in rats. Male and female rats were exposed for 1 year to levels of 50 or 40 mg/m^3 of antimony trioxide for 7 hours/day, 5 days/week and were subsequently observed for 1 year. Neoplastic lesions developed in female rats; non-neoplastic lesions developed in the males. Data are summarized in Table 5-2. The Federal Register (1983b) states that the Watt (1981) and Wong (1979) studies provided evidence of oncogenic effects in female rats, but finds the studies inadequate for prediction of or assessment of risk. In the Watt (1981) study, only one sex was used. In the Wong et al. (1979) study, only one exposure level was used. No tumors developed in rats exposed occasionally (1 hour every 2 months for 1 year) by inhalation to antimony trioxide at 1700 mg/m^3 (Vorwald, 1963). No additional experimental details are available (U.S. EPA, 1978b).

Davies (1973) carried out a retrospective epidemiological study in 1081 antimony process workers, including workers studied previously by Oliver (1933), McCallum (1963, 1967) and McCallum et al. (1971). The purpose of this study was to assess the incidence of lung cancer. Incidences of cancer death rates were higher when compared with expected community lung cancer death rates. The study was initiated in 1962 after a man who had processed antimony died of lung cancer. Seven other deaths from lung cancer were

TABLE 5-1

Tumor Incidence in Female Rats Exposed by Inhalation to Antimony Trioxide[a,b]

Dose (mg/kg/day)	Duration of Treatment	Duration of Study	Target Organ	Tumor Type	Tumor Incidence (p value)
0.24	365 days	730 days	lung	carcinoma	15/17 (NR)
0.09				adenoma	1/17 (NR)
0					1/13 (NR)

QUALITY OF EVIDENCE

Weaknesses of study:	Duration of exposure, latency cannot be determined; only one sex used; non-specific tumors in both control and experimental animals (liver and mammary); no array of dose-response levels; lack of adequate control of exposure levels.

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[a] Source: ASARCO, Inc., 1980; Watt, 1981, 1983

[b] Purity of compound not reported

NR = Not reported

TABLE 5-2

Tumor Incidence in Male and Female Rats Exposed by Inhalation to Antimony Trioxide[a,b]

Dose	Sex	Duration of Treatment	Duration of Study	Target Organ	Tumor Type	Tumor Incidence (p value)
50 mg/m³ 3.3 mg/kg/day	M	365 days	730 days	lung	non-neoplastic lesions	NR
40 mg/m³ 2.64 mg/kg/day	F			lung	tumor	NR

QUALITY OF EVIDENCE

Weaknesses of study: Only one dose used, no array of dose-response levels. Lack of adequate control of exposure levels.

---

[a] Source: Wong et al., 1979

[b] Purity of compound not reported

NR = Not reported

found retrospectively among workers in the previous 8 years. Of the 1081 men and women studied for the period of 1961-1971, 937 people were alive and living in England, 20 had emigrated, 56 had died and 68 were difficult to trace. Of the 56 deaths, 10 were due to lung cancer. Of those who died of lung cancer, 9/10 had worked solely at antimony smelting and related activities. The expected rate of death was calculated to be 5.7. In the 45-64 age group, 8/9 lung cancer deaths were reported where the expected rate was 4.5. Additional data were gathered in 1973, but necessary details were lacking (NIOSH, 1978). These data also showed increased incidences, 18 vs. an expected 10.25 in the smelter subgroup and 13 vs. 7.22 in the 45-64 age group. Data are summarized in Table 5-3.

Another retrospective epidemiology study is in process at Asarco, Inc. (Federal Register, 1983a). This study is a dual study of health effects from exposure to both antimony and lead in workers in a lead smelter in East Helena, MT. The lead contains low levels of antimony, with exposure levels averaging 0.01-0.1 mg/m^3 (3-8 hours of exposure) with some exposures as high as 2.08 mg/m^3. Two lung cancer deaths of a total of 81 deaths occurred in a population of 437 males (working at least 1 year between January 1, 1947 to December 31, 1975). The expected deaths due to cancer were 3.4. Four deaths also occurred from non-neoplastic respiratory disease, compared with an expected 2.9 deaths from this effect. Data are summarized in Table 5-4.

5.2. MUTAGENICITY

Positive effects were observed in a recombination-repair-deficient _Bacillus_ _subtilis_ assay (Kanematsu et al., 1980). In the rec-assay procedure, antimony trioxide inhibited cellular growth more strongly in a recombinant-deficient strain of _B_. _subtilis_ (M45) than in the wild type (H17).

TABLE 5-3

Incidence of Lung Cancer in Humans Exposed by Inhalation to Antimony Trioxide[a]

Size of Exposed Population	Size of Population	Sex	Level of Exposure	Duration of Exposure	Number of Deaths Observed	Number of Deaths Expected	Relative Risk (p value)
1081 men and women employees	unknown community	NR[b]	NR	8 years	10/56 due to lung cancer	5.7/56	1.75 (NR)

QUALITY OF EVIDENCE

Weakness of study: Retrospective epidemiology study

Comments: Reference #14, NIOSH (1978) - only abstract of report available

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[a] Source: Davies, 1973

[b] Probably male, since 9/10 worked in antimony smelting

NR = Not reported

TABLE 5-4

Incidence of Lung Cancer in Humans (males) Exposed by Inhalation to Antimony Trioxide[a]

Size of Exposed Population	Size of Population	Level of Exposure	Duration of Exposure	Number of Deaths Observed	Number of Deaths Expected	Relative Risk (p value)
437	unknown	0.01-0.1 mg/m^3 for 3-8 hours	unknown employees worked between 1946 and 1970	2/437[b]	3.4/437	<1

QUALITY OF EVIDENCE

Weakness of study: Retrospective epidemiology study; ASARCO lead smelter in East Helena, MT.

Comments: Incidence in this population less than expected.

---

[a] Source: Federal Register, 1983a

[b] A total of 81/437 deaths were reported overall for this population.

These results suggest DNA-damaging effects with antimony trioxide. Results with the Salmonella typhimurium TA1535, TA100, TA98, TA1537 and TA1538 were negative (Federal Register, 1979). Paton and Allison (1972) reported on the induction of chromosomal aberrations in cultured human cells. Metal salts (antimony sodium tartrate) were added to leukocyte cultures in a concentration of 2.3×10^{-9} molar and the number of cells with chromatid breaks were statistically increased ($p<0.05$).

5.3. TERATOGENICITY

Belyaeva (1967) exposed 24 female rats repeatedly to antimony trioxide dust by inhalation for 1.5-2 months at doses of 250 mg/m^3 for 4 hours/day (NIOSH, 1978). Rats were treated in this manner 3-5 days before estrus and then mated. Exposure to the dust was continued until 3-5 days prior to delivery. Of the treated rats, 16/24 conceived whereas all of the control rats (10) became pregnant. Average litter sizes for treated rats were 6.2 and for control rats 8.3. No morphological changes were seen in the fetuses. Resorption and fetal deaths were not discussed. No fetal abnormalities were observed when 30 animals were exposed to single doses of metallic antimony (50 mg/kg i.p.). Results are summarized in Table 5-5. Casals (1972) administered a solution of antimony dextran glycoside (RL-712) intramuscularly, containing 125 or 250 mg. Sb/kg (~50 mg Sb/kg bw) to pregnant rats and mice on five occasions between the 8th and 15th day of gestation. No fetal changes were seen.

5.4. OTHER REPRODUCTIVE EFFECTS

In an acute experiment by Belyaeva (1967), antimony metal dust (diameters up to 5 μ) was administered in a single i.p. dose of 50 mg/kg to 30 female rats. The animals were mated while in estrus, 3-5 days after treatment. A total of 15 became pregnant; 8 during the first mating, 5 during the second mating (1 month post-treatment) and 2 during the third mating.

TABLE 5-5

Effects of Acute and Chronic Exposures to Antimony on the Litter Sizes and Weights of Newborn Rats*

Exposure	No. of Females	Mean Weight of Dams (g)	Mean Litter Size	Weight of Infants at Birth (g)	Weight of Infants After 3 Weeks (g)
Single i.p. Metallic Antimony					
Exposed	30	190.0±5.2	5.4±0.2	5.5±1.1	32±9.2
Control	12	188.5±2.5	7.8±0.5	5.4±0.5	28.1±0.4
Repeated Inhalation Antimony Trioxide					
Exposed	24	195.5±7.2	6.2±1.0	5.6±1.2	25.2±2.8
Control	10	197.8±14.0	8.3±0.2	5.5±1.6	23.5±3.4

*Source: Belyaeva, 1967

The remaining 15 rats failed to conceive, although 6 of them were mated once, 7 were mated twice and 3 were mated three times. In the control group, only one rat failed to become pregnant after one mating. Experimental animals that did become pregnant produced fewer offspring/animal when compared with control animals.

Microscopic studies of 23 rats (from both the acute and repeated exposure groups) showed uterine and ovarian changes. Ovarian follicles either lacked ova or contained uncharacteristic ova. Cortical hyperemia of the ovaries, follicular cysts and metaplasia of the uterus were seen. Some animals also exhibited tubular metaplasia. Effects were more severe in the animals receiving intraperitoneal metallic antimony.

Belyaeva (1967) compared women working in an antimony metallurgical plant with a similar group of women not exposed to antimony. Extensive gynecologic examinations were conducted twice a year from 1962-1964. The exposed group had higher incidences of spontaneous abortions (12.5 vs. 4.1%), premature births (3.4 vs. 1.2%) and gynecologic problems (77.5 vs. 56%). The gynecologic problems included menstrual cycle disorders (61.2 vs. 35.7%), inflammatory disease (30.4 vs. 55.3%) and other reproductive problems (8.4% of exposed women). Antimony concentrations in the blood of exposed workers were 10 times greater than controls. Urine levels of antimony ranged from 2.1-2.9 mg/100 mℓ. Analysis of biologic fluids detected antimony breast milk concentrations of 3.3 ± 2 mg/ℓ; placental tissue, 3.2-12.6 mg/100 mℓ; amniotic fluid, 6.2 ± 2.8 mg/100 mℓ; and umbilical cord blood, 6.3 ± 3 mg/100 mℓ. These women were exposed to unspecified amounts of antimony trioxide, metallic antimony and antimony pentasulfides.

Children born to these exposed women weighed significantly less after 1 year when compared with children born to nonexposed women; there were no statistically significant differences in weights at birth of the two sets of children.

Aiello (1955) observed a higher rate of premature deliveries and frequent dysmenorrhea among female workers engaged in antimony smelting and processing (U.S. EPA, 1980). Bradley and Frederick (1941) noted frequent abortions in rabbits given repeated high oral doses of metallic antimony (Federal Register, 1979).

5.5. SUBCHRONIC AND CHRONIC TOXICITY

5.5.1. Effects on Animals. Smyth and Carpenter (1948) gave five male and five female rats antimony trioxide at doses ranging from 60-1070 mg/kg bw/day in the diet for 30 days. No experimental controls were used. Reduced growth and appetites and unspecified micropathology in liver, kidney, spleen or testes were reported in the highest dose group. No effects were seen at 270 mg/kg/day. Gross et al. (1955a) gave 20 rats and rabbits 2% antimony trioxide in their diet for 8 months. The only adverse effect reported was slower weight gain in treated animals compared with controls. In spite of high thyroid levels of antimony, no abnormalities or endocrine disturbances were noted. No gross or microscopic abnormalities of other organs or tissues were noted (NIOSH, 1978).

Bradley and Frederick (1941) administered daily doses of antimony metal in feed to rats. Doses of 1000 mg/kg bw were fed for up to 12 months. Consistent injury to the heart and an increase in connective and fibrous tissue in the myocardium were observed. Myocardial congestion with engorgement of cardiac blood vessels and dilatation of the right heart were seen at the higher dose levels. Moderate periportal congestion was noted; spleen hyperplasia and mild glomular nephritis were also observed. Antimony oxides produced no splenic changes (NIOSH, 1978). Bradley and Frederick (1941) also administered antimony _ad libitum_ in feed to rabbits. Daily doses of 40 mg of metallic antimony for 4 months were used. In 10 rabbits and 20 rats, antimony was retained in the heart, liver, kidney, lungs and bone. In rats, only small amounts were retained at an average of 1 mg/carcass, indicating little storage of this substance. In spectrographic determinations, high antimony retention corresponded to predominant pathological findings. No other information was available.

Sunagawa (1981) fed rats 1.0 and 2.0% Sb_2O_3 for 24 weeks. At 2% Sb_2O_3, red cell counts were significantly decreased and white cell counts were decreased at 1%. α-Glutamyl transpeptidase and alkaline phosphatase levels were elevated. Antimony levels after ingestion of 1.0% antimony trioxide were: blood, 148.11 ppm; spleen, 61.88 ppm; lung, 28.84 ppm; and liver, 15.19 ppm. At the 2.0% ingestion level, antimony levels were: blood, 114.87 ppm; spleen 55.71 ppm; lung, 27.26; and liver, 17.74 ppm. Gross et al. (1955a) administered 2% antimony trioxide to a group of 20 rats (sex not specified) for ~240 days. Although the rats fed antimony gained weight at a slower rate than pair-fed control animals, no gross or microscopic pathologic effects were noted.

Schroeder et al. (1970) supplied Long-Evans rats with drinking water containing 5 ppm antimony as potassium antimony tartrate. Experimental groups consisted of 52 male control rats, 54 female control rats, 51 male rats given antimony and 59 female rats given antimony. Rats were fed a diet consisting of: seed rye flour (60%), dry skim milk (30%), corn oil (9%), iodized sodium chloride (1%) with added vitamins and ferrous sulfate. The drinking water for both the control and test groups contained 50 ppm zinc, 10 ppm manganese, 5 ppm copper, 1 ppm trivalent chromium, 1 ppm cobalt and 1 ppm molybdenum. Rats were monitored for up to 45 months, or until the death of the last survivor.

Animals were weighed weekly until 6 weeks old and subsequently at monthly intervals. Animals dying during the course of the experiment were necropsied and heart, lung, kidney, liver and spleen examined for histopathological changes. In addition, blood chemistries and urinalysis were performed; however, it is not clear at what point in the study these values were obtained.

Animals exposed to antimony showed a significant decrease in longevity ($p<0.001$). Longevity was defined as the mean survival time for the last 10% of the surviving animals. This reduction was apparent for both males and females. Fasting glucose values in males and females were not significantly different than controls. However, nonfasting glucose values were significantly reduced for both sexes (i.e., in the antimony-treated rats glucose values did not rise in the fed state and in fact appeared to be lower). Although cholesterol levels were significantly different, the male values were higher and the female values were lower.

In a study of similar design (utilizing Swiss mice), lifespan in females appeared to be adversely affected. The authors did not report statistical analyses in this study which was designed primarily to evaluate carcinogenic potential (Kanisawa and Schroeder, 1969).

Watt (1983) exposed female CDF rats and S-1 miniature swine to antimony trioxide dust at 1.6 ($\pm$1.5) mg/m^3 (as Sb) or 4.2$\pm$(3.2) mg/m^3 (as Sb) for 6 hours/day, 5 days/week for 1 year, and observed rats for an additional year post-exposure. No adverse effects were observed in miniature swine.

These doses were close to the TLV. In rats, lung toxicity was observed and total lung weights were elevated. Body weights were significantly higher for exposed rats. Exposed lungs appeared mottled and both neoplastic and non-neoplastic responses were present; incidence and severity increased with level and length of exposure. The non-neoplastic response occurred in both high-dose and low-dose rats and consisted of focal fibrosis, adenomateous hyperplasia, multinucleated giant cells, cholesterol clefts, pneumocyte hyperplasia and pigmented macrophages.

Fairhall and Hyslop (1947) concluded that antimony trioxide dust was much more toxic than ingested antimony trioxide. Experiments showed that inhalation continued for long periods of time produced definite cell injury.

Gross et al. (1952) administered antimony trioxide by inhalation and intratracheal injection, causing endogenous lipoid pneumonia. Inhalation exposure was 100-125 mg/m^3 for 25 hours/week for 14.5 months.

Gross et al. (1955b) exposed 50 rats to antimony trioxide dust by inhalation in doses of 100-125 mg/m^3 for 100 hours/month for 14.5 months. Nine rats died from pneumonia (18%). No controls were used and time of death was not specified. Fatty degeneration of alveolar macrophages with lipid accumulation in the alveoli was observed along with lung fibrosis. The average particle size inhaled was 0.6 µ. Considerable deposits of antimony trioxide were observed in lymph nodes, but no fibrosis of these structures was observed. Gross et al. (1955b) also exposed 20 rabbits to 100-125 mg/m^3 of antimony trioxide for 100 hours/month for 10 months. Seventeen rabbits died from pneumonia (85%). Controls were not used and time of death was not specified. Adverse effects seen in rabbits were the same as those noted in rats. There was more pronounced interstitial pneumonia in rabbits, and less diffuse fibrosis.

Cooper et al. (1968) exposed two groups of animals, each composed of 10 male and 10 female Sprague-Dawley rats (84 days old; males weighed 342-425 g, females weighed 227-276 g) to antimony by inhalation. A dosage of 1700 mg/m^3 for 1 hour, 1-6 times every 2 months was used. Animals were exposed to either antimony trioxide or powdered ore; exposure was for 66-311 days with the trioxide and 66-366 days with the powdered ore. Animals from each group were sacrificed periodically and examined. No experimental controls were used. Immediately after the first exposure, a transient and mild edema and lung congestion was seen with the powdered ore. A phagocytic response was seen in the lungs in both groups of animals 66 days after the first exposure. As time of exposure increased, spleens exhibited scattered particles with moderate reticuloendothelial proliferation. No hepatic or renal pathology was noted.

Dernehl et al. (1945) exposed 24 guinea pigs to antimony trioxide by inhalation at a dosage of 45.4 mg/m^3. Animals were exposed initially for 2 hours/day, 7 days/week for 3 weeks and subsequently for 3 hours/day for the rest of the experiment; total time of exposure ranged from 33-609 hours (27 weeks). The particle size inhaled was <1 μ. Retention in the lung was 50%, producing a theoretical daily retention of 1.6 mg. All 24 animals showed microscopic evidence of pneumonitis (retaining a total of 13-424 mg) and four animals died, two from pneumonia. All animals retaining ≥50 mg of antimony trioxide showed scattered subpleural hemorrhages (NIOSH, 1978). In 11/15 animals exposed for ≥138 hours (6 days) (a retention of 77 mg in the lungs), fatty degeneration of the liver was observed. Half of these animals also exhibited hyperplasia in lymphoid follicles of the spleen with an abnormal amount of blood pigments present. Exposed animals also had decreased white blood cell counts. It was noted that the adrenal cortex was also affected (U.S. EPA, 1981).

Belyaeva (1967) exposed rats to antimony trioxide by inhalation at doses of 250 mg/m^3 for 4 hours/day for 1.5-2 months. Microscopic examination of 23 rats revealed uterine and ovarian changes such as absence of ova or uncharacteristic ova, cortical hyperemia of the ovaries, follicular cysts and uterine and tubular metaplasia. Without describing the changes, the author stated that "substantial pathological changes were observed in lungs, liver, kidneys and pancreas" (Newman et al., 1981).

Gudzovskii (1968) reported experiments with rats inhaling 50 mg/m^3 antimony trioxide for 2-4 hours/day for 6 months. Lungs were studied histologically. Regardless of type of compound used, deep morphological changes in lung parenchyma and respiratory tract walls were seen, with development of focal endogenic lipoidal pneumonia.

In a pilot experiment, Potkonjak and Vishnjich (1983) injected 0.5 ml (50 mg of dust) of antimony oxide (Sb_2O_3 and Sb_2O_5) intraperitoneally and endotracheally into two groups of female Wistar rats (250 g), 12 animals/group. The animals were sacrificed after 2 months and lung and omentum were examined histologically. Lungs showed small whitish nodules; similar formations were seen in omentum. Histologically, small heaps of coniocytes contained phagocytized particles with some fibrous proliferation, which the authors concluded was pneumoconiosis of a noncollagenous nature.

Antimony trioxide was administered to rats subcutaneously 5 times/week for 3 months for a total dose of 165 mg (Minkina et al., 1973). No changes were observed after four injections, but a broadening of the cortical layer of the adrenal cortex was seen after 20 injections, due primarily to growth of the fosicular and retriculate zones, with cell size in these zones increased. Signs of increased adrenocortical functional activity were present until after 35 or 47 injections, at which times these signs disappeared.

The authors considered this a habituation of the adrenals to a toxic substance. Effects in animals are summarized in Table 5-6.

5.5.2. Effects on Humans. The Romans used antimony-rich alloys in wine cups, so that sufficient antimony leached from the cup induced vomiting (Browning, 1969). In the 1700s, chemists who inhaled fumes from antimony and glass workers who pulverized the substance to make colored glass had neurological and pulmonary problems (Ramazzini, 1713). McWalter (1910) suggested that neuritis suffered by printers was due to antimony; this was also noted by Schrumpf and Zabel (1910) as a similar problem in typesetters. Seitz (1923) noted blood abnormalities due to antimony exposure of workers in a type foundry and described results of studies in cats, rabbits and guinea pigs that suggested that antimony was responsible for the blood changes. Feil (1939) described adverse dermal and mucous membrane effects among workers engaged in producing antimony metal, antimony trioxide and antimony sulfide, with the ore having concentrations of 10 mg/kg. Fourteen of the 15 foundry workers had active skin eruptions or histories of skin eruptions along with conjunctivitis, tracheitis, pharyngitis and headache. Six anemias were also reported. The author also noted that workers exposed to antimony sulfides did not exhibit signs of toxicity.

Klucik and Ulrich (1960) found changes in EKG readings including T-wave depression borderline normal QT waves, prolonged atrio-ventricular transmission, prolonged QRS complexes and ST segment in metal workers exposed to antimony trisulfide and trioxide. Forty-two metal workers were exposed to antimony trisulfide and trioxide at concentrations ranging from 1.3-237 g/m^3 of inhaled air (concentrations reported may have a 1000 times error and may actually have been 1.3-237 mg/m^3). The average age of the workers was middle to late 40's. There are some doubts about this study, due to

TABLE 5-6

Effects of Antimony Oxides and Antimony in Animals*

Compound	Species	Dose or Exposure	Effects	Reference
Antimony trioxide	rat	1 or 2% in diet for 24 weeks	decreased red and white counts, enzyme effects	Sunagawa, 1981
Antimony trioxide	rat	1070 mg/kg bw/day for 1 month in diet	reduced growth; histological pathology liver, kidney, spleen or testes	Smyth and Carpenter, 1948
		270 mg/kg/day for 1 month in diet	no effect	
Antimony metal	rat	1000 mg/kg for 12 months in feed	heart injury, liver congestion, spleen hyperplasia, glomular nephritis	Bradley and Frederick, 1941
Antimony trioxide	rat	1.6 (±1.5) or 4.2 (±3.2) mg/m^3 for 6 hours/day, 5 days/week for 1 year	lung fibrosis and hyperplasia at both doses	Watt, 1983
Antimony trioxide	rat	100-125 mg/m^3, 25 hour/week for 14.5 months	lipoid pneumonia	Gross et al., 1952
Antimony trioxide	rat	1700 mg/m^3 for 1 hour every 2 months for 66-311 days	acute pulmonary congestion, phagocytic response	Cooper et al., 1968
Antimony trioxide	rat	250 mg/m^3 for 4 hours/day, 7 days/week for 1.5-2 months	fewer offspring; lung, liver, kidney and pancreatic changes	Belyaeva, 1967
Antimony trioxide	rat	50 mg/m^3 for 2-4 hours/day for 6 months	lipoidal pneumonia, deep morphological changes in the lung	Gudzovskii, 1968
Antimony trioxide	mice	dose not stated; inhalation	mild lung fibrosis persisting for 6-9 months	Hsin et al., 1981
Antimony trioxide	guinea pig	45.4 mg/m^3 for 33-609 hours (27 weeks)	pneumonitis, subpleural hemorrhage	Dernehl et al., 1945
Antimony trioxide	guinea pig	45.4 mg/m^3 for 138-609 hours	liver degeneration, splenic damage, blood alterations	Dernehl et al., 1945

*Source: NIOSH 1978

statistical problems, data gathering problems and the probability that arsenic was also present in the plant.

McCallum (1963, 1967) and McCallum et al. (1971) studying antimony pneumoconiosis in workers in an antimony processing plant could identify and quantify antimony lung deposits in cases not exhibiting overt signs. It was possible to establish associations between years of employment, amount of antimony in the lungs and severity of pneumoconiosis. Antimony concentrations in the work environment were >0.5 mg/m³, with the trioxide dust particles being <1 μ in diameter. During tapping operations, concentrations of antimony averaged 37 mg/m³ while other parts of the factory had concentrations up to 5 mg/m³. Pneumoconiosis was diagnosed by radiographic examination in an unreported number of workers, most exhibiting no overt symptoms. Dermatitis and "antimony" spots were observed and were most prevalent during the work week, often disappearing on weekends and holidays. Samples of urine taken from three workers with pneumoconiosis had antimony concentrations of 425, 480 and 680 μg/ℓ. In a retired worker, urinary concentrations were 55 μg/ℓ after 7 months of retirement and 28 μg/ℓ after 4 years of retirement. Of the men employed at the Newcastle-upon-Tyne antimony works, 44/262 had pneumoconiosis ascribed to antimony. Results of microscopic examination of a worker who had died from lung carcinoma were also reported. Antimony dust laden macrophages in alveolar septa and perivascular tissues were described (McCallum, 1963).

A new method of x-ray analysis was used in screening 113 workers, most of whom were employed for <20 years and worked at different operations for varying periods (McCallum et al., 1971). Concentrations of lung antimony ranged from undectectable to 11 mg/cm² lung area. A man with the highest level of antimony in his lungs (11.2 mg/cm²) had been employed 35 years, 16 in packing antimony trioxide.

Renes (1953) investigated 78 workers in the United States engaged in smelting antimony sulfide ore. Average concentrations of antimony in plant air was 10.8 mg/m^3 (arsenic was 0.73 mg/m^3). The author postulated that antimony and arsenic were present as oxides because of the nature of the smelting operation. During 5 months of plant operation, 69 workers reported illnesses such as respiratory irritation and dermatitis. Heavily-exposed workers had pneumonitis. Abdominal cramps, diarrhea, vomiting, dizziness, headaches, nerve tenderness and tingling and prostration were observed in 9 other heavily-exposed workers. Seven men had antimony excreted in the urine at concentrations ranging from traces to 60 mg/100 cc.

LeGall (1969) reported that pneumoconiosis occurred in 10/40 persons working with furnaces used to produce antimony trioxide, with workplace concentrations ranging from 0.3-14.7 mg/m^3; particle sizes were <3 μ.

Karajovik (1958) studied 101 workers at an antimony smelter in Yugoslavia. Work environment dust was found to contain 36-90% antimony trioxide, 1-6% antimony pentoxide, 0.3-9% arsenic trioxide, 2-4% ferric oxide, 1-12% silica, and 1-7% free silicon dioxide; particle sizes were <0.5 μ. As revealed by x-rays, 14/62 workers had pneumoconiosis. Twenty-two workers had emphysema with bronchitis and were <40 years of age. Upper respiratory tract catarrhal symptoms were observed in 51 workers. Conjunctivitis was seen in 12 workers, and 16 workers had dermatitis.

Three workers out of 28 antimony smelter workers were found to have pneumoconiosis (Cooper et al., 1968). These workers had exposure durations of 1-15 years to antimony sulfide and antimony trioxide. Air concentrations ranged from 0.081-138 mg/m^3 but in most cases were $\leq$10 mg/m^3. Particle size was not specified. Oliver (1933) examined six men involved in antimony trioxide production (Newcastle-upon-Tyne, England). Slight anisocytosis was seen in one individual, blood pressures were below average and antimony excretion was noted in feces but not urine.

Potkonjak and Pavlovich (1983) reported occupational exposure, clinical findings, lung function and radiological characteristics of 51 workers (ages 31-54, mean 45.23 years) in an antimony smelting plant (worked 9-31 years, mean 17.91 years), who were exposed to airborne dust containing up to 88% antimony trioxide and the remainder, antimony pentoxide. Pneumoconiotic changes were seen in lungs after 1 decade of employment. No systemic changes were seen except for "antimony dermatosis." No massive lung fibrosis was noted. Diffuse, densely distributed puntate opacities, diameter <1 mm, concentrated in mid-lung were found. Enlarged dense hilar shadows and emphysematous changes were seen. Chronic coughing (60.8%), conjunctivitis (27.5%), antimony dermatosis (32/51), upper airway inflammation (35.3%), chronic bronchitis (37.3%) chronic emphysema (34.5%) and pleural adhesions (27.3%) were observed. No malignant lesions were noted. In comparison, McCallum (1963) found that 8.5% of 268 subjects had pneumoconiosis and McCallum (1967) and McCallum et al. (1971) established a relationship between years of employment at an antimony trioxide plant, the amount of antimony found in workers lungs and the severity of pneumoconiosis observed.

Anzon Limited, in the United Kingdom, is conducting an epidemiology study of 2104 male workers engaged in production of antimony substances and zircon in the Anzon Limited works at Newcastle-upon-Tyne. This study, started in 1961, is investigating chronic health effects. Since 1961, 98/1651 workers have died. The U.S. EPA has requested information on causes of death, vocational history and smoking habits (Federal Register, 1983b).

In hot, moist environments, skin irritation and rashes are seen with exposure to antimony trioxide. Stevenson (1965) found that 23/150 workers at an antimony factory in Newcastle had this condition. A worker who had been exposed to fireproof packing material containing antimony trioxide had dermatitis (Paschoud, 1964). Linch and Sigmund (1976), however, after conducting patch tests with antimony trioxide containing 0.29% arsenic on 45 women and 7 men (ages 17-35), found no skin reactions. Thivolet et al. (1971) reported dermatitis in two workers producing an antimony-trioxide based powder for treating internal parasites in chickens. The dermatitis was periodic, as described in other studies.

Much information has also been accumulated on the effects of treatment of parasitic diseases with organic compounds containing antimony (Browning, 1969; NIOSH, 1978). Effects of occupational exposure to antimony oxides are summarized in Table 5-7.

5.6. OTHER RELEVANT INFORMATION

Flury (1927) reported on the effects of various oral doses of Sb_2O_3 and Sb_2O_5 on rats, cats and dogs, but insufficient information was available to calculate dosages. Also, the number of test animals was inadequate, and only changes in body weight were monitored (Newman et al., 1981). Rats were unaffected by 2000 mg/day and dogs by 400 mg/day of Sb_2O_3. Oral doses of 150 mg in cats caused GI distress (Newman et al., 1981). A study by Smyth and Carpenter (1948) indicated that rats ingesting up to 270 mg/kg doses of Sb_2O_3 for 30 days showed no effects. The smallest daily dose that produced symptoms (reduced growth, appetite and histological changes in liver, kidney, spleen and testes) in rats was 1070 mg/kg Sb_2O_3.

TABLE 5-7

Effects of Exposure to Antimony Oxides on Humans[a]

Compound	Concentration mg/m^3	Duration	Effects	Reference
Antimony trioxide	0.40-70.7 (particle size <1 μ)	<6 months	skin, mucous membrane irritation, respiratory illness, GI disturbance	Renes, 1953
Antimony trioxide, pentaoxide	16-248 (particle size <0.5 μ)	NR	skin and mucous membrane irritation	Karajovic, 1958
Antimony trioxide	<0.5 (particle size <1 μ)	NR	pneumoconiosis, skin irritation	McCallum, 1963
Antimony trioxide	0.081-138	1-15 years	pneumoconiosis	Cooper et al., 1968
Antimony trioxide	NR (particle size <1 μ)	6 months to 35 years	pneumoconiosis	McCallum et al., 1971
Antimony trioxide	NR	NR	possible increased risk of lung cancer	Davies, 1973
Antimony trioxide	NR	2-13 years	mild dermatitis, slight anisocytosis	Oliver, 1933
Antimony trioxide	0.3-56 (particle size 0.57-1.46 μ)	NR	no effects	Linch and Sigmund, 1976
Antimony trioxide	patch test	NR	dermatitis	Paschoud, 1964
Antimony trioxide	patch test	NR	dermatitis if skin scarified	Thivolet et al., 1971
Antimony trioxide	patch test	NR	no effects	Linch and Sigmund, 1976

[a] Source: NIOSH, 1978

A maximum dose (oral) of 275 mg/kg of Sb_2O_3 administered daily to rats caused no effect (Newman et al., 1981). Westrick (1953) investigated effects of antimony on thyroid function in rats and rabbits. Rats were given a dose of Sb_2O_3 (2% by weight) added to their diet for 49 days. Rabbits were given doses of 13 mg/kg Sb_2O_3 each day for 21 days. No difference in body weight or oxygen consumption in either species was observed.

Acute poisoning from antimony trioxide was reported by Dunn (1928) and Monier-Williams (1934). Seventy people who drank lemonade from preparations left overnight in white enamelware buckets (the enamel contained 2.88% antimony trioxide) became ill. Antimony trioxide had been leached from the enamel by the acidic lemonade. Fifty-six people were hospitalized, suffering from burning stomach pains, colic, nausea and vomiting; most recovered within 3 hours. Analysis found that the lemonade contained 0.013% metallic antimony. Each person ingesting ~300 mℓ lemonade would have received 36 mg antimony, which is similar to an emetic dose listed in the British Pharmacopoeia. Werrin (1963) reported a similar incident in 150 children at a church picnic where pink lemonade was served from enamelware containers. The children recovered in a few hours.

Skin irritation from antimony compounds has been studied by Oliver (1933), Stevenson (1965), Paschoud (1964), Linch and Sigmund (1976), LeGall (1969) and Cooper et al. (1968). Pneumonitis from antimony has been investigated by Renes (1953), LeGall (1969), McCallum (1963), Karajovik (1958) and Cooper et al. (1968). Oliver (1933) found below average blood pressure and continuous excretion of antimony in feces, but not urine, in antimony oxide smelter workers.

Antimony oxide LD_{50} values are summarized in Table 5-8. Bradley and Frederick (1941) determined that the minimum lethal i.p. dose of antimony trioxide in rats (total of 149 animals in study) was 325 mg/100 g bw. LD_{50}s were also determined for metallic antimony (10 mg/100 g bw) and antimony pentoxide (400 mg/100 g bw). Myocardial congestion and death due to myocardial failure occurred. Livers showed moderate periportal congestion with vacuolization, degeneration and polymorphonuclear infiltration. Oxides and sulfides had no effect on the spleen but metallic antimony caused moderate hyperplasia with eosinophilia. A mild glomular nephritis also occurred with oxides and sulfides and was toxic with antimony potassium tartrate. Cellular changes were seen in myocardial fibers; other cells were edematous. Livers showed subcapsular congestion, blood pigmentation, and cloudy and finely reticulated cytoplasm. Antimony was also administered intraperitoneally to guinea pigs (88 animals) and LD_{50} of 15.0 mg/100 g bw was determined. Pathological findings were similar to those observed in with rats.

Gross et al. (1955a) administered a single i.p. injection of 20 mg of antimony trioxide to three rats. On examination 2 months later, some peritoneal adhesions were seen. Antimony dust deposits were encapsulated into the peritoneal wall by a thin layer of connective tissue. One year later the adhesions were absent and only small foci remained. Liver, kidney, spleen and intestinal walls were normal.

Gross et al. (1955a) administered one i.v. injection of 20 mg antimony trioxide to rabbits or repeated injections totaling 200 mg (53 mg/kg) at 2-day intervals; the animals were observed for up to 1 year. No abnormalities were noted in the organs of either group. Gross et al. (1955a) administered single doses of antimony trioxide of 20 mg i.v. to rabbits.

TABLE 5-8

LD_{50} of Antimony Oxides and Metallic Antimony

Compound	Species	Route	Dose (mg/kg)	Reference
Antimony trioxide	rat	i.p.	2250	Bradley and Frederick, 1941
Antimony pentoxide	rat	i.p.	4000	Bradley and Frederick, 1941
Antimony metal	rat guinea pig	i.p.	100 150	Bradley and Frederick, 1941
Antimony metal	rat	i.p.	7000	Bradley and Frederick, 1941
Antimony metal	rat	i.p.	110-130	JRB Associates, Inc., 1980

No abnormalities were noted. Another group of rabbits received repeated i.v. injections totaling 200 mg (53 mg/kg) at 2-day intervals and were observed for 1 year. No abnormalities were noted. Subcutaneous injections of 0.089 mg/kg Sb_2O_3 administered 25 times within 1 month to rats had little effect on body weight, organ health, oxygen consumption and brain function (Levina and Chekunova, 1965). The only effects were vascular distention and small hemorrhages in parenchymateous organs. Levina and Chekunova (1965) found that no mortality resulted from s.c. injections of up to 50 mg/kg Sb_2O_3 in rats, mice and rabbits.

Baseline values of antimony concentrations in blood, urine and feces of normal, healthy Japanese city dwellers were measured by Hirayama (1959). Antimony concentrations in the blood of 104 subjects ranged from undetectable to 13.5 μg antimony/100 g whole blood, with an overall median value of 2.4 μg/100 g. For men, the median value was 2.5 μg; for women, 2.1 μg/100 g. Urine values (24-hour) were determined for 80 men and 55 women, ranging from undetectable to 18.5 μg/day. Overall median values were 5.3 μg/day. For men, the median value was 5.4; for women, 5.1 μg/day. Antimony excreted in feces of 85 men and 41 women ranged from a trace to 56 μg/day; half were $\leq$13 μg/day. The author concluded that these baselines were common to industrial cities.

Lung distribution of antimony was studied by Molokhia and Smith (1969) in male and female subjects ranging in age from 40-70 years. A mean wet-weight concentration of 0.095 ppm was determined, with a range of 0.007-0.45 ppm and a median concentration of 0.056 ppm.

6. AQUATIC TOXICITY

Aquatic toxicity data regarding antimony trioxide are available; however, none were located regarding the pentoxide as cited in the Appendix.

6.1. ACUTE TOXICITY

The U.S. EPA (1978a) and Buccafusco et al. (1981) reported toxicity tests of antimony trioxide in bluegill sunfish, Lepomis macrochirus, at levels up to 530 mg/ℓ for 96 hours. Mortality at the test concentrations did not reach 50%; therefore, the LC_{50} value for all test durations was >530 mg/ℓ. The MATC for the bluegill was 530 mg/ℓ. Curtis and Ward (1981) reported that the 96-hour LC_{50} of antimony trioxide to fathead minnows, Pimephales promelas, was >833 mg/ℓ since no significant mortality occurred below this level. Tarzwell and Henderson (1960) exposed fathead minnows to antimony trioxide at levels up to 80 mg/ℓ in hard and soft water but reported no mortality.

In the marine sheepshead minnow, Cyprinodon variegatus, LC_{50} values for 24, 48, 72 and 96 hours of exposure were all between 6.22 and 8.30 mg/ℓ. It was reported, however, that the solubility of antimony trioxide in seawater was near 5.0 mg/ℓ. The MATC was 6.22 mg/ℓ for C. variegatus (Heitmuller et al., 1981). In the euryhaline mummichog, Fundulus heteroclitus, Dorfman (1977) reported that all 24-, 48-, 72- and 96-hour LC_{50} values were >1000 mg antimony trioxide in tests conducted at 6.0 and 21.6 ppt salinity.

Toxicity tests in two crustaceans, Daphnia magna (freshwater) and Mysidopsis bahia (marine), were conducted by the U.S. EPA (1978a). The 24- and 48-hour LC_{50} value for D. magna was >530 mg/ℓ for both exposure durations and the NOEC value was 530 mg/ℓ. In the marine shrimp, M. bahia, the LC_{50} for exposures up to 96 hours was >4.15 mg/ℓ. The low solubility of antimony trioxide in seawater (5.0 mg/ℓ) prevented tests at higher concentrations. The NOEC in the marine shrimp was 0.83 mg/ℓ.

6.2. CHRONIC TOXICITY

The U.S. EPA (1978a) conducted chronic flow-through tests using fathead minnows, *Pimephales promelas*, to determine a MATC for antimony trioxide. No effect on survival or health of fish was noted at the highest concentration tested (0.0075 mg/ℓ); therefore, the MATC value was >7.5 ppb.

In an unpublished manuscript, Kimball (n.d.) reported 28-day threshold levels of 4.5, 1.1 and 2.3 μg Sb/ℓ for survival, growth in length and growth in weight, respectively. The estimated MATC values based on these threshold levels were 6.5, 1.6 and 3.2 μg/ℓ, respectively.

Kimball (n.d.) also reported a 28-day LC_{50} of 4.51 μg Sb/ℓ in *Daphnia* after exposure to antimony trioxide. The MATC for reproduction was 5.42 μg/ℓ, and the highest concentration that produced no toxic effects during the 28-day test was 4.16 μg Sb/ℓ.

6.3. PLANT EFFECTS

The 24-, 48-, 72- and 96-hour EC_{50} values for decreased growth of the freshwater green alga, *Selenastrum capricornutum*, were >0.83, 0.61, 0.61 and 0.63 μg/ℓ. The NOEC for antimony trioxide was 0.17 mg/ℓ (U.S. EPA, 1978a). In the marine algae, *Skeletonema costatum*, EC_{50} values for intervals up to 96 hours were all >4.15 mg/ℓ. Antimony trioxide was insoluble in seawater at concentrations >5.0 mg/ℓ. The NOEC for *S. costatum* was reported to be 4.15 mg/ℓ.

6.4. RESIDUE

During bioconcentration tests of antimony trioxide, fish were exposed at 0.0033 mg/ℓ for 28 days (U.S. EPA, 1978a). No antimony was detected in the tissues of exposed fish, so that a BCF of 0 was reported.

6.5. OTHER RELEVANT INFORMATION

Pertinent data regarding the toxicity of antimony oxides to aquatic organisms were not located in the available literature as cited in the Appendix.

7. EXISTING GUIDELINES AND STANDARDS

7.1. HUMAN

NIOSH (1978) recommended a criteria for antimony of 0.5 mg/m^3. A TLV of 0.5 mg/m^3 for doses of antimony trioxide has been recommended by ACGIH (1983).

OSHA adopted the ACGIH (1983) TLV for antimony of 0.5 mg/m^3 as the Federal standard (Code of Federal Regulations, 1981). Finland, the Federal Republic of Germany, the Democratic Republic of Germany, Rumania, the USSR, Sweden and Yugoslavia had adopted the same exposure limits (U.S. EPA, 1980b).

The U.S. EPA (1981) states that

> "if compliance with the OSHA standard for antimony and its compounds is absolute, the maximal occupational exposure would equal 0.03 mg/kg/day."

Calculation of this exposure is based on the assumption that all exposure is in the trioxide form and absorption of the inhaled compound is 100%, and is also based on the rat inhalation data from Asarco, Inc, where positive oncogenic effects were observed in rats exposed to 4.2 mg/m^3 antimony trioxide 6 hours/day, 5 days/week for 1 year. The OSHA standard is one-seventh of the rat dosage that caused lung lesions. The safety range for this standard appears narrow, due to problems extrapolating from rats to humans, and is a cause for concern, since 68% of samples taken from a survey done by Donaldson and Cassady (1979) exceeded the OSHA standard (U.S. EPA, 1981).

An ADI of 287 μg/day for a 70 kg human has been calculated (U.S. EPA, 1980). This value was based on a 5 ppm exposure level of antimony as potassium antimony tartrate in drinking water that had no effect on the rate of growth of rats or mice, but produced significant reductions in the lifespans of both species (Kanisawa and Schroeder, 1969; Schroeder et al., 1970).

This was used as a LOEL to calculate the ADI, using an uncertainty factor of 100. The corresponding ambient water quality criterion is 145 μg/ℓ.

The USSR has a drinking water limit of 50 μg/ℓ for antimony. The WHO has not developed a guideline for antimony (Federal Register, 1983a).

7.2. AQUATIC

Guidelines and standards specifically for the protection of aquatic organisms from the toxic effects of antimony oxides were not located in the available literature as cited in the Appendix. The U.S. EPA (1980) did not recommend a specific criterion to protect aquatic life from antimony compounds in general, but stated that concentrations as low as 1.6 and 0.61 caused toxic effects in freshwater organisms (chronic exposures) and algae, respectively. More sensitive species would require lower antimony levels to prevent toxic effects.

8. RISK ASSESSMENT

A large number of studies exist with animals and humans on various antimony compounds (NIOSH, 1978), and the great majority show no carcinogenic effects. These studies are considered inadequate by today's testing standards for detecting carcinogenic activity (Federal Register, 1979). The ITC recommended that metallic antimony, antimony trisulfides and antimony trioxide be tested for carcinogenicity, based on studies of workers exposed to antimony compounds, who may be at increased risk to lung cancer (U.S. EPA, 1979b; Davies, 1973).

After release of the ITC report, a submission under TSCA section 8(e) was made available by ASARCO, Inc. This study by Watt (1980) demonstrated formation of non-neoplastic (fibrotic) and neoplastic lesions (lung tumors) in female rats (15/17) after 1 year of observation following 1 year of exposure (6 hours/day, 5 days/week) to antimony trioxide. Dosage level reported by ASARCO, Inc., that caused tumors was 4.2 mg/m³. A lower dosage exposure of 1.6 mg/m³ did not cause tumors (U.S. EPA, 1980). The dosages reported in the Federal Register (1983a) were 1.6 (±1.5) mg/m³ (non-neoplastic) and 4.2 (±3.2) mg/m³ (neoplastic). According to the Federal Register (1983a), another TSCA section 8(e) submission was presented by ASARCO, Inc., describing results of histopathology studies confirming the preliminary diagnosis of non-neoplastic and neoplastic lesions in female rats exposed to antimony trioxide. This study was further described in a thesis abstract by Watt (1983).

Another report has been submitted to TSCA from Midwest Research Institute describing a study in which both sexes of rats were exposed to levels of "50±40 mg/m³" antimony trioxide for 1 year (7 hours/day, 5 days/week) and then were held for 1 year of observation (Wong et al., 1979; EPL, 1981).

The histopathology report confirmed development of neoplastic lesions in female rats exposed to antimony trioxide (EPL, 1982). Male rats developed non-neoplastic, but no neoplastic, lesions from exposure to antimony trioxide. No other information is available regarding this experiment. The U.S. EPA finds neither of these studies adequate for determination of oncogenic risk (Federal Register, 1983a). Lack of adequate control of exposure levels in both experiments makes their use unacceptable. No supportive data on carcinogenic risk from metallic antimony exist and, in addition, the U.S. EPA states that there would be no significant difference in exposure because of oxidation to antimony trioxide during processing (Federal Register, 1983a). Because of these considerations, derivation of a q_1^* value is not appropriate.

Teratogenic effects were discussed by Belyaeva (1967), Bradley and Frederick (1941), Casals (1972) and James et al. (1966). After ITC review, the U.S. EPA considers these reports not to be a basis for finding that antimony substances may present an unreasonable risk of teratogenic effects (Federal Register, 1983a).

Belyaeva (1967) reported other reproductive effects in women chronically exposed to antimony substances and also performed experiments with female rats to evaluate potential reproductive effects (see Section 5.4.). The exposure level used in these animal studies was 250 mg/m³, which is ~500 times higher than the current OSHA recommended TLV of 0.5 mg/m³ for antimony substances. In addition, it is 150 times higher than the mean level that produced non-neoplastic respiratory lesions in male rats (Watt, 1980). In the Belyaeva (1967) study, there were also serious inadequacies in protocol, such as questionable controls, lack of measurement of exposure levels in the human studies, and use of only one sex and one dose in the animal studies. Therefore, the U.S. EPA found only a very weak basis for antimony substances causing a risk of reproductive effects. Protection against neoplastic and non-neoplastic respiratory injury would also protect against risk of reproductive effects (Federal Register, 1983a). The U.S. EPA does not consider the data from Kanematsu et al. (1980) and from Paton and Allison (1972) adequate for assessing the mutagenicity of antimony trioxide because of differences in physical and chemical properties (such as solubility in water) between sodium antimony tartrate and antimony oxides (Federal Register, 1983b).

While the above studies raise concern for the potential carcinogenic, teratogenic, and reproductive effects of antimony oxides, the concerns are not sufficient to preclude the derivation of an ADI, although additional studies on these effects would be desirable.

For the purposes of deriving an ADI for antimony oxides, the three most relevant studies are those of Smyth and Carpenter (1948), Sunagawa (1981) and Gross et al. (1955a). As summarized in Chapter 5, Smyth and Carpenter (1948) administered antimony trioxide in the diet for 30 days to groups of five male and female rats at doses ranging from 60-1070 mg/kg bw/day. At the highest dose level, animals had reduced appetite and growth, along with unspecified pathologic changes in the liver, kidney, spleen or testes. At the dose of 270 mg/kg bw/day, no effects were seen. Because of the limited experimental detail provided by this study, it cannot be determined if the unspecified pathological lesions were associated with decreased food consumption.

In the study by Sunagawa (1981), rats were administered antimony trioxide in the diet at levels of 1.0% (10,000 mg/kg diet) and 2.0% (20,000 mg/kg diet) for 24 weeks. Assuming that the food consumption of rats is equivalent to 5% of their body weight, these levels resulted in average daily doses of 500 and 1000 mg/kg bw, respectively. Based on the information contained in the abstract of this Japanese study, neither dose level resulted in overt toxic effects in terms of general appearance and behavior. At the highest dose level, erythrocytes were decreased significantly and increases were seen in serum glutamic-oxalacetic transaminase, γ-glutamyl transpeptidase and alkaline phosphastase. At the lower dose level, a decrease was noted in white blood cells and an increase was noted in γ-glutamyl transpeptidase. No results of tissue pathology or organ weights were reported.

Bradley and Frederick (1941) fed rats antimony potassium tartrate and metallic antimony at levels equivalent to daily doses of 100 and 1000 mg/kg, respectively, for up to 1 year. They reported significant myocardial damage in both dose groups.

In the study by Gross et al. (1955a), 20 rats were administered 2% antimony trioxide in the diet for ~240 days. Although little experimental detail is provided, the only effect noted was decreased weight gain when compared with pair-fed controls. Gross and microscopic pathologic examinations revealed no treatment-related changes.

The data on antimony trioxide can be compared with the studies by Kanisawa and Schroeder (1969) and Schroeder et al. (1970), in which groups of 50 male and female rats were given potassium antimony tartrate in drinking water at 5 mg/ℓ for up to 39 months (Schroeder et al., 1970) and groups of 54 male and female mice were given potassium antimony tartrate in the drinking water at 5 mg/ℓ for 540 days (~18 months) (Kanisawa and Schroeder, 1969). In both mice and rats, lifespans were reduced significantly. Assuming that drinking water consumption in rats is equivalent to

7.8% of body weight, the daily dose to rats in the study by Schroeder et al. (1970) is 0.39 mg/kg bw.

As summarized in Chapter 7, the U.S. EPA (1980) has used the LOEL of 0.39 mg/kg/day from Schroeder et al. (1970) with an uncertainty factor of 100 to derive an ADI for antimony of 287 µg/day for a 70 kg human or 4.1 µg/kg/day. The substantially higher doses of antimony trioxide tolerated by rats in the studies by Sunagawa (1981) and Gross et al. (1955a) and supported by the shorter term study of Smyth and Carpenter (1948) could suggest a NOAEL of 500 mg/kg/day for antimony trioxide. However, these studies were of inadequate duration to reveal adverse effects on longevity reported by Schroeder et al. (1970) for antimony potassium tartrate. Although one might suspect that the poorly soluble oxides would be less readily absorbed than the tartrate; data are inadequate for quantitative comparisons of absorption efficiency.

Although antimony has been used for treatment of human patients for schistosomiasis, the route of treatment has generally been i.m. or i.v. Fairhall and Hyslop (1947) reported that antimony is less toxic following i.v. administration than following oral exposure. These investigators stated that 150 mg may be fatal following oral administration, whereas 30-150 mg was recommended for i.v. treatment. The reason for this observation is not clear, but it does cast further doubt on the applicability of the limited clinical data to estimation of an oral ADI.

Until more complete data are available concerning the absorption and effects of antimony oxides, it seems prudent to utilize the Schroeder et al. (1970) data as the basis for ADI estimation. These data suggest the lowest effect level for antimony and reflect the only study conducted for the entire lifespan of the experimental animals. In addition, a group of people who consumed estimated doses of 36 mg of antimony trioxide (~0.43 mg/kg as antimony) experienced acute GI distress. This dose is considerably lower than the NOELs determined in the subchronic antimony trioxide studies.

Schroeder et al. (1970) estimated that in their experimental animals the drinking water level of 5 ppm antimony corresponded to a daily dose of 350 µg/kg for rats, the most sensitive species. At this dose level, animals of both sexes showed significantly decreased longevity combined with alterations in blood glucose and cholesterol levels. Therefore, 350 µg/kg/day corresponded to a LOAEL in this study. Applying an uncertainty factor of 1000 (10 for interspecies extrapolation, 10 for interindividual variability and 10 to estimate a NOAEL from a LOAEL) results in an ADI of 0.35 µg/kg/day or 24.5 µg/day antimony for a 70 kg man. Correcting for differences in molecular weights, the respective ADIs for antimony trioxide, tetroxide and pentoxide are 29.3, 30.9 and 32.5 µg/day. When additional data become available concerning the toxicology of antimony oxides it would be advisable to review these estimates.

9. REPORTABLE QUANTITY

9.1. REPORTABLE QUANTITY (RQ) RANKING BASED ON CHRONIC TOXICITY

Composite scores have been calculated for a number of oral and inhalation studies. These data are summarized in Table 9-1. In the oral studies, the largest CSs occurred with the studies by Smyth and Carpenter (1948) and Bradley and Frederick (1941), but these studies are subchronic, poorly reported and not recent. The studies by Sunagawa (1981) are also of interest, but again, experimental details are not available for an accurate assessment of risk. Finally, the U.S. EPA (1983) indicated that antimony oxide may be more toxic by inhalation than by ingestion.

In the inhalation studies, the largest CS value is from the recent studies by Watt (1983). A previous RQ value (U.S. EPA, 1983) had been determined from the studies by Gross et al. (1952, 1955a). These authors observed similar dose-dependent histologic changes in the lungs of both rats and rabbits exposed to 89 or 100-125 mg/m³ of antimony trioxide for 25 hours/week for 10-14.5 months. Using the lowest exposure levels, a MED for humans of 132 mg/day was calculated. In this calculation, the exposure for 25 hours/week was expanded to a continuous exposure of 168 hours/week, with the assumption of an animal breathing volume of 224 m³ and divided by the assumed weight for a rat of 0.35 kg. This MED corresponds to an RV_d of 2.5 and an RV_e of 6 for the changes in lung histology and the occurrence of lipoid pneumonia. Because of reported deaths due to pneumonia, the RV_e value was increased to 8, since this is death due to impairment of the lung function. Using this rationale, a CS of 20 was obtained, corresponding to an RQ classification of 1000.

TABLE 9-1

Composite Scores for Antimony Trioxide

Species	Route	Dose/Exposure (mg/kg/day)	Chronic Human MED (mg/day)	RV$_d$	Effect	RV$_e$	CS	RQ	Reference
Rat	oral	1000	598.5	1.3	Heart injury; liver, spleen, kidney damage	7	9.3	1000	Bradley and Frederick, 1941
Rat	oral	1070	64.0*	2.8	Pathology of liver, kidney, spleen, testes	7	19.5	100	Smyth and Carpenter, 1948
Rat	oral	1000	598.5	1.3	Decrease in body weight	4	5.2	5000	Gross et al., 1955a
Rat	oral	500	29.9*	3.3	Changes in enzyme and blood parameters	2	6.6	1000	Sunagawa, 1981
Rabbit	oral	40	1.44*	5.3	Cardiomyopathy	7	36.8	100	Bradley and Frederick, 1941
Rat	inhalation	0.49	101.6	2.5	Lipoid pneumonia, histologic changes in lungs	8	20.0	1000	Gross et al., 1952, 1955a
Rat	inhalation	26.7	319.3*	3.2	Lung, liver, kidney, pancreatic changes; uterine and ovarian changes	6	19.5	1000	Belyaeva, 1967
Rat	inhalation	5.3	6.4*	4.3	Lipoidal pneumonia, changes in lung morphology	4	17.2	1000	Gudzovskii, 1968
Rat	inhalation	10	2.2	5.0	Fibrotic changes in lungs	6	30.0	100	Watt, 1983
Guinea pig	inhalation	1.2	15.4	3.7	Pneumonitis, subpleural hemorrhage, splenic lymphoid hyperplasia (small number of animals used)	6	22.3	1000	Dernehl et al., 1945
Human	inhalation	0.122	8.6	4.1	Pneumoconiosis	3	12.3	1000	McCallum, 1963

*This value was obtained by dividing by a factor of 10 after determination of the value because a subchronic study was used for calculation.

The occupational studies of McCallum (1963) were used as supportive evidence for the Gross et al. (1952; 1955a) data. A conservative exposure level of 0.5 mg Sb/m³ was chosen, corresponding to an antimony trioxide level of 1.2 mg/m³ and resulting in an MED of 8.6 mg/day when exposure was expanded to a 7-day week. A breathing volume of 10 m³ was assumed. The MED of 8.6 mg/day corresponds to a RV_d of 4.1 and an RV_e of 3 for the pneumoconiosis revealed upon radiologic examination of the workers who did not complain of breathing difficulty. The CS obtained is 12.3, corresponding to an RQ of 1000.

The studies by Watt (1983) were performed with dosage levels similar to those extrapolated from the McCallum (1963) study, and produced effects similar to those observed with chronic exposure in humans. In the Watt (1983) study, the lower dosage level of 1.6 mg/m³ produced fibrotic changes in the lungs of both sexes of rats. Exposure to this dosage level was for 6 hours/day, 5 days/week for 1 year, which converts to a dosage of 0.18 mg/kg/day. This exposure regimen was used to calculate a chronic human MED of 2.19 mg/day, which corresponds to an RV_d value of 5.0 and an RV_e value of 6 for fibrotic changes in the lungs. These values result in a CS of 30.0, corresponding to an RQ of 100. It should be noted that the higher dosage levels used in the Watt (1983) experiments caused lung tumors in female rats at a dosage of 4.2 mg/m³ for 6 hours/day, 5 days/week for 1 year. These data are summarized in Table 9-2.

9.2. WEIGHT OF EVIDENCE AND POTENCY FACTOR ($F=1/ED_{10}$) FOR CARCINOGENICITY

The U.S. EPA (Federal Register, 1983a) reports that none of the available studies, including the Watt (1983), Wong et al. (1979) and Belyaeva (1967) studies, are suitable for determination of carcinogenic risk. The mutagenicity studies are not conclusive; the retrospective epidemiology studies are still in progress.

TABLE 9-2

Antimony Oxides

Minimum Effective Dose (MED) and Reportable Quantity (RQ)

Route:	inhalation
Dose*:	2.19 mg/day
Effect:	fibrotic lung changes
Reference:	Watt, 1983
RV_d:	5.0
RV_e:	6
Composite Score:	30.0
RQ:	100

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*Equivalent human dose

Some data exist regarding induction of lung tumors in female rats in a chronic study by Watt (1983), using dose levels close to the present OSHA standard. Fibrotic lung changes have been seen repeatedly in human workers exposed chronically to antimony compounds, with increased incidence in workers associated with smelting operations. Therefore, limited evidence may exist for carcinogenicity, with data from rats and humans placing this chemical in IARC Group 2B.

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APPENDIX

LITERATURE SEARCHED

This profile is based on data identified by computerized literature searches of:

CA SEARCH (Files 308, 309, 310, 311, 320
TOXLINE
RTECS
OHM TADS
STORET
SRC Environmental Fate Data Bases
SANSS
AQUIRE
TSCAPP

These searches were conducted in December, 1984. In addition, hand searches were made of Chemical Abstracts (Collective Indices 6 and 7), and the following secondary sources were reviewed:

ACGIH (American Conference of Governmental Industrial Hygienists). 1980. Documentation of the Threshold Limit Values, 4th ed. (Includes Supplemental Documentation, 1981, 1982, 1983). Cincinnati, OH. 486 p.

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Worthing, C.R. and S.B. Walker, Ed. 1983. The Pesticide Manual. British Crop Protection Council. 695 p.

In addition, approximately 30 compendia of aquatic toxicity data were reviewed, including the following:

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